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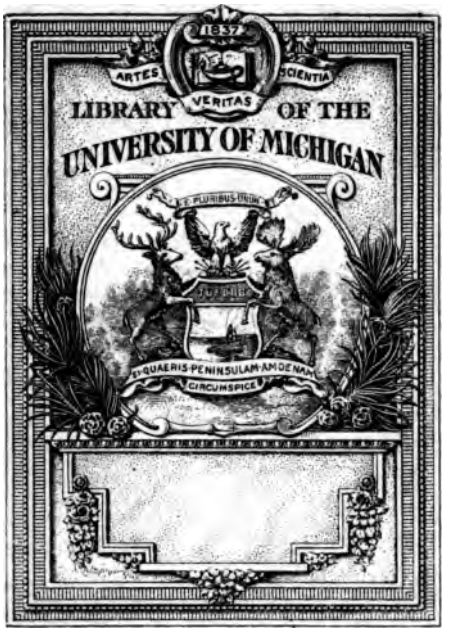
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VOLUME THE FIFTY-SEVENTH.

LONDON:
LONGMANS, GREEN, READER, AND DYER,
PATERNOSTER ROW.

1874.

PRINTED BY J. E. ADLARD, BARTHOLOMEW CLOSE.

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FOR THE SESSION OF 1874-75.

BARNES, ROBERT, M.D.
BARWELL, RICHARD.
BEALE, LIONEL S., M.B., F.R.S.
CHOLMELEY, WILLIAM, M.D.
CHURCH, WILLIAM SELBY, M.D.
CLARK, FREDERICK LE GROS, F.R.S.
FAGGE, CHARLES HILTON, M.D.
FERGUSSON, SIR WILLIAM, Bart., F.R.S.
FOX, WILSON, M.D., F.R.S.
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HOLTHOUSE, CARSTEN.
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RADCLIFFE, CHARLES BLAND, M.D.
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SMITH, THOMAS.
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WILKS, SAMUEL, M.D., F.R.S.
WOOD, JOHN, F.R.S.

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1805. WILLIAM SAUNDERS, M.D.
1808. MATTHEW BAILLIE, M.D.
1810. SIR HENRY HALFORD, Bart., M.D., G.C.H.
1813. SIR GILBERT BLANE, Bart., M.D.
1815. HENRY CLINE.
1817. WILLIAM BABINGTON, M.D.
1819. SIR ASTLEY PASTON COOPER, Bart., K.C.H., D.C.L.
1821. JOHN COOKE, M.D.
1823. JOHN ABERNETHY.
1825. GEORGE BIRKBECK, M.D.
1827. BENJAMIN TRAVERS.
1829. PETER MARK ROGET, M.D.
1831. SIR WILLIAM LAWRENCE, Bart.
1833. JOHN ELLIOTSON, M.D.
1835. HENRY EARLE.
1837. RICHARD BRIGHT, M.D., D.C.L.
1839. SIR BENJAMIN COLLINS BRODIE, Bart., D.C.L.
1841. ROBERT WILLIAMS, M.D.
1843. EDWARD STANLEY.
1845. WILLIAM FREDERICK CHAMBERS, M.D., K.C.H.
1847. JAMES MONCRIEFF ARNOTT.
1849. THOMAS ADDISON, M.D.
1851. JOSEPH HODGSON.
1853. JAMES COPLAND, M.D.
1855. CÆSAR HENRY HAWKINS.
1857. SIR CHARLES LOCOCK, Bart., M.D.
1859. FREDERIC CARPENTER SKEY.
1861. BENJAMIN GUY BABINGTON, M.D.
1863. RICHARD PARTRIDGE.
1865. SIR JAMES ALDERSON, M.D., D.C.L.
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1871. THOMAS BLIZARD CURLING.
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The figures succeeding the words *Trans.* and *Pro.* show the number of Papers which have been contributed to the Transactions or Proceedings by the Fellow to whose name they are annexed. *Sci. Com.* is attached to the names of those who have served on the Scientific Committees of the Society.

OCTOBER, 1874.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *Abercrombie, John, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1851 *Acland, Henry Wentworth, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 Acosta, Elisha, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1842 †ACTON, WILLIAM, 17, Harley street, Cavendish square. *Trans.* 1.

1852 ADAMS, WILLIAM, Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 2.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 38, Park square, Leeds. *Trans.* 3.

1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 18, Bryanston street, Portman square. *Trans.* 2.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1870 ARNOTT, HENRY, Assistant-Surgeon to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 6, Nottingham place, Marylebone road.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly, and 21, Hyde park place, Cumberland gate. C. 1821, 1830-31.

Elected

1851 ASHTON, THOMAS JOHN, Consulting Surgeon to the St. Marylebone Infirmary; 20, Park square east, Regent's park.

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Anatomy and Physiology at, St. Bartholomew's Hospital; 26, Wimpole street, Cavendish square. *Trans.* 2.

1869 BAKEWELL, ROBERT HALL, M.D., Dunedin, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Principal Medical Officer, Gibraltar. [6, Whitehall yard.] C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 7, Compton terrace, Islington. C. 1872. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.

1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.

1862 BARKER, EDGAR, 21, Hyde park street.

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

Elected

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. Thomas's Hospital; Examiner in Midwifery at the University of London; 31, Grosvenor street. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Anatomy and Clinical Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 2.

1844 BASHAM, WILLIAM RICHARD, M.D., Senior Physician to the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. T. 1871. *Trans.* 2.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. * *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1836 BEAUMONT, WILLIAM RAWLINGS, Consulting Surgeon to the Toronto General Hospital; Toronto, Canada West. *Trans.* 3.

1871 BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell. [Hanwell.]

1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

Elected

1871 BELLAMY, EDWARD, Senior Assistant-Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon to the Royal Infirmary for Children and Women, Waterloo road; 59, Margaret street, Cavendish square.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1845 BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.

1854 BIRD, PETER HINCKES, F.L.S., 4, Clifton Terrace, Lytham, Lancashire.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.

1851 BIRKETT, JOHN, F.L.S., *Treasurer*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; Examiner in Surgery at the University of London; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-74. *Trans.* 8. *Sci. Com.*

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

1843 †BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.

Elected

1840 BLAKISTON, PEYTON, M.D., F.R.S. [55, Victoria street, Pimlico.]

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.

1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2. V.P. 1870-71. *Sci. Com.*

1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]

1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.

1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 1.

Elected

1851 BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 56, Curzon street, Mayfair. *Trans.* 1.

1844 †BROOKE, CHARLES, M.A., F.R.S., Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72.

1857 *BROWN, ROBERT, late Surgeon to the Cumberland Infirmary, Wintersh, Brampton.

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., late Professor of Physiology and Pathology, Massachusetts Medical College, Harvard University, Boston, U.S.; Rue Gay-Lussac, 28, Paris. *Sci. Com.*

1867 BRUNJES, MARTIN, 42, Brook street, Grosvenor square.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Casualty Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica at the University of London; 23, Somerset street, Portman square.

1860 BRYANT, THOMAS, Surgeon to Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Trans.* 8; *Pro.* 1. *Sci. Com.*

1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.

Elected

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., President of the Royal College of Physicians; Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Trans.* 2.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.

1873 BUTLIN, HENRY TRENTHAM, Surgical Registrar to St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square.

1871 BUTT, WILLIAM F., 12, South street, Park lane.

1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Joint-Lecturer on Surgery at, St. Bartholomew's Hospital; Lecturer on Anatomy and Physiology at the Royal College of Surgeons; Examiner in Anatomy at the University of London; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.*

1852 *CANNEY, GEORGE, M.D., Bishop-Auckland, Darlington, Durham.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 69, Wimpole street, Cavendish square, W. *Trans.* 1.

Elected

1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*

1868 CAVAFY, JOHN, M.D., Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; Physician to the Victoria Hospital for Children; 13, Arlington street, Piccadilly.

1871 CAYLEY, WILLIAM, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the London Fever Hospital; 58, Welbeck street, Cavendish square.

1845 CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 24, Mount street, Grosvenor square. C. 1861. V.P. 1867. L. 1869-72. *Trans.* 1.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1837 †CHAPMAN, HENRY THOMAS, 13, Cradock street, Swansea, Glamorgan. C. 1858.

1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

1873 *CHISHOLM, EDWIN, Camden, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.

Elected

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Assistant-Physician to, and Lecturer on Comparative Anatomy at, St. Bartholomew's Hospital; 2, Upper George street, Bryanston square.

1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 14, St. Thomas's street, Southwark, and Lee, Kent. S. 1847-9. V.P. 1855-6. *Trans.* 5.

1848 CLARKE, JOHN, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; Physician to the General Lying-in Hospital; 52, Queen Anne street, Cavendish square. C. 1866.

1866 CLARKE, WILLIAM FAIRLIE, M.A. Oxon., Assistant-Surgeon to the Charing Cross Hospital; 12, Mansfield street, Cavendish square. *Trans.* 2.

1861 *CLARKE, WILLIAM JAMES, Surgeon to the Huddersfield Infirmary; John-William street, Huddersfield, Yorkshire.

1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.

1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.

1857 COATES, CHARLES, F.R.C.P. Edinb., Physician to the Bath United General Hospital; 10, Circus, Bath.

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 7, Suffolk place, Pall mall. *Trans.* 2.

Elected

1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.

1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.

1865 COOPER, ALFRED, Surgeon to the Royal Hospital for Diseases of the Chest, Additional Surgeon for Out-patients to the Lock Hospital; Assistant-Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1819 COOPER, GEORGE, Brentford, Middlesex.

1873 COOPER, GEORGE HENRY CRESSWELL, F.R.C.S. Ed.; 35, Compton terrace, Highbury.

1841 †COOPER, GEORGE LEWIS, one of the Surgeons to the National Vaccine Institution, and Teacher of Vaccination to the Medical School of University College; Surgeon to the Bloomsbury Dispensary; 7, Woburn place, Russell square. C. 1860-1. *Trans.* 1.

1843 †COOPER, WILLIAM WHITE, *Vice-President*, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.-P. 1873-4.

1868 CORNISH, WILLIAM ROBERT, Surgeon, Madras Army; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 9, Clarendon place, Belfast.

1839 *CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

1853 CORY, WILLIAM GILLETT, M.D., 47, Rue Tour Notre Dame, Boulogne-sur-Mer.

1847 †COTTON, RICHARD PAYNE, M.D., Physician to the Hospital for Consumption, Brompton; 33, Cavendish square. C. 1863.

Elected

1828 †COULSON, WILLIAM, F.L.S., Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 29, St. James's place.

1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.

1873 CREIGHTON, CHARLES, M.B., 2, Grosvenor villas, Wimbledon.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square.

1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.

1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.

1860 CROSS, RICHARD, M.D., 5, Queen street, Scarborough.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

Elected

1849 *Crowfoot, William Edward, Beccles, Suffolk.

1851 Cumming, James Cameron, M.D.

1846 Curling, Henry, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 †Curling, Thomas Blizard, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1873 Curnow, John, M.D., Professor of Anatomy at King's College, London; 3, Warwick street, Cockspur street.

1847 Currey, John Edmund, M.D., Lismore, County Waterford.

1822 Cusack, Christopher John, Chateau d'Eu, France.

1852 Cutler, Thomas, M.D., Spa, Belgium.

1872 Dalby, William Bartlett, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 1.

1836 *Daniel, James Stock, Ramsgate, Kent.

1848 Daubeny, Henry, M.D., San Remo, Italy.

1846 Davies, Frederick, M.D., 124, Gower street, Bedford square. C. 1873.

1853 Davies, Robert Coker Nash, Rye, Sussex.

1852 Davies, William, M.D., 18, Gay street, Bath.

1852 Davis, John Hall, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; 24, Harley street, Cavendish square. C. 1869-70.

1818 Dawson, James, Wray Castle, Windermere.

1867 Day, William Henry, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1867 De Meric, Victor, Surgeon to the Royal Free Hospital, and to the German Hospital, Dalston; 52, Brook street, Grosvenor square.

Elected

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; Physician to the Hospital for Sick Children; Examiner at the Royal College of Physicians, and at the University of Cambridge; 11, Chesterfield street, Mayfair. C. 1874. *Trans.* 12. *Sci. Com.*

1844 DICKSON, ROBERT, M.D., F.L.S., Cambridge Lodge, Harmondsworth, Slough. C. 1860.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting-Ophthalmic Surgeon to the Asylum for Idiots; 29, Lower Seymour street, Portman square. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.

1845 DODD, JOHN.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1853 DRUITT, ROBERT, F.R.C.P. *Trans.* 2.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 99, Southampton row, Russell square.

1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1845 DUFFIN, EDWARD WILLSON, 18, Devonshire street, Portland place. *Trans.* 1.

1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.

Elected

1871 *DUKES, CLEMENT, M.B. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 82, Brook street, Grosvenor square. *Trans.* 5. *Sci. Com.*

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 DYER, HENRY SUMNER, M.D., Sennowe Hall, Guist, Norfolk. C. 1854-5.

1872 EAGER, REGINALD, M.D., Superintendent at St. Luke's Hospital for Lunatics, Old street.

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B., Lond. Surgeon-Accoucheur to the Western General Dispensary; 5, Albion place, Hyde park square.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.

1848 ELLIS, GEORGE VINER, Professor of Anatomy in University College, London. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., Belle Grove Villa, Welling.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

Elected

1842 †ERICHSEN, JOHN ERIC, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.

1845 EVANS, WILLIAM JULIAN, M.D., Pinner, Middlesex.

1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; and Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 4.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., Lynton, North Devon.

1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

1862 FARQUHARSON, ROBERT, M.D., Lecturer on Materia Medica at St. Mary's Hospital; Physician to the Belgrave Hospital for Children; 23, Brook street, Grosvenor square.

1844 FARRE, ARTHUR, M.D., F.R.S., Physician-Accoucheur to H.R.H. the Princess of Wales; 12, Hertford street, Mayfair. C. 1857. V.P. 1864. *Sci. Com.*

1872 FAYRER, JOSEPH, C.S.I., M.D., F.R.S. Ed., Honorary Physician to H.M. the Queen, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-Major, Bengal Army; Member of the Indian Medical Board; 16, Granville place, Portman square.

1872 FENWICK, JOHN C. J., M.D., 30, Devonshire street, Portland place.

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to, and Lecturer on Histology at, the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.

1841 †FERGUSSON, SIR WILLIAM, Bart., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon to King's College Hospital; 16, George street, Hanover square. C. 1849-50, V.P. 1863-4. *Trans.* 4.

Elected

1852 *Field, Alfred George, Alverton Manor House, Stratford-on-Avon.

1849 Fincham, George Tupman, M.D., Physician to the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1866 Fish, John Crockett, B.A., M.B. Camb., Junior Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1836 †Fisher, Sir John William, 33, Park Lane. C. 1843-4.

1860 Fitzgerald, Thomas George, Surgeon-Major; 6, Whitehall yard.

1866 Fitzpatrick, Thomas, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

1842 Fletcher, Thomas Bell Elcock, M.D., Consulting Physician to the Birmingham General Hospital; 7, Waterloo street, Birmingham. *Trans.* 1.

1864 *Folker, William Henry, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1848 †Forbes, John Gregory, 82, Oxford Terrace, Hyde Park. C. 1868-9. *Trans.* 3.

1852 †Forster, John Cooper, *Secretary*, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 29, Upper Grosvenor street. C. 1868-9. S. 1873-4. *Pro.* 1.

1865 Foster, Balthazar Walter, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 Fox, Edward Long, M.B., Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.

1858 Fox, Wilson, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the Unisity of London; 67, Grosvenor street. *Trans.* 3.

Elected

1871 FRANK, PHILIP, M.D., Cannes, France.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 29, Spring gardens.

1836 †FRENCH, JOHN GEORGE, late Surgeon to the St. James's Infirmary; 10, Cunningham place, Maida hill. C. 1852-3.

1849 FRERE, ROBERT TEMPLE, M.A., F.R.C.P., 143, Harley street.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark.

1865 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital, 16, Connaught square, Hyde park. *Trans.* 2.

1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.

1867 GARLIKE, THOMAS W., 126, Tulse hill, Brixton.

1854 GARROD, ALFRED BARING, M.D., F.R.S., late Professor of Materia Medica in King's College, London, and Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.

1857 GASCOYEN, GEORGE GREEN, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. S. 1866-9. C. 1871-2. *Trans.* 3. *Sci. Com.* 2.

1851 GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park.

1819 GAULTER, HENRY.

1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874.

Elected

1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to St. Bartholomew's Hospital; Assistant-Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Trans.* 1.

1821 *GEORGE, RICHARD FRANCIS, 20, Marlborough buildings, Bath.

1870 GODSON, CLEMENT, M.D., C.M., Physician to the Samaritan Free Hospital; 8, Upper Brook street, Grosvenor square.

1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; 4, Highbury park. C. 1864-5. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 1.

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury Square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; 74, Wimpole street, Cavendish Square.

1843 †GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. *Trans.* 3.

Elected

1868 GRIGG, WILLIAM CHAPMAN, M.D., Medical Officer, Queen Charlotte's Lying-in-Hospital; Assistant Obstetric Physician to the Westminster Hospital; 6, Curzon street, Mayfair.

1814 GROVE, JOHN, M.D., Salisbury.

1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris.

1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., *Vice-President*, Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Trans.* 4.

1837 GULLY, JAMES MANBY, M.D., Great Malvern, Worcestershire.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.

1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

1848 HALLEY, ALEXANDER, M.D., F.G.S., 16, Harley street, Cavendish square.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 76, Harley street, Cavendish square. C. 1851. V.P. 1869.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; 13, Sussex place, Onslow gardens.

1836 HARDING, JOHN FOSSE, Mount Sandford, Southborough, Tunbridge Wells. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.

Elected

1857 HARLEY, GEORGE, M.D., F.R.S., 25, Harley street, Cavendish square. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.

1864 HARLEY, JOHN, M.D., F.L.S., Assistant-Physician to, and Joint Lecturer on Physiology at, St. Thomas's Hospital; 78, Upper Berkeley street, Portman square. *Trans.* 6.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., Physician to St. Bartholomew's Hospital; 24, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.

1841 †HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear and to the Freemasons' Female Charity; Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1854 HAVILAND, ALFRED, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.

1870 HAWARD, J. WARRINGTON, Assistant-Surgeon to the Hospital for Sick Children; 5, Montagu street, Portman square. *Trans.* 1.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 6, Brook street, Grosvenor square.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square.

Elected

1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.

1848 *HEALE, JAMES NEWTON, M.D., Winchester, Hants.

1865 HEATH, CHRISTOPHER, Surgeon to University College Hospital, and Lecturer on Operative Surgery in University College, London; 9, Cavendish place, Cavendish square.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1829 †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1843 HEWETT, PRESCOTT GARDNER, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans.* 7. *Sci. Com.*

1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; Examiner in Midwifery at the University of London; 36, Berkeley square.

1872 HEYN, JULIUS CHARLES WILLIAM, M.D., 88, Lange Voorhout, the Hague, Holland.

1875 HIGGENS, CHARLES, F.R.C.S, Assistant Ophthalmic Surgeon to Guy's Hospital; 38, Brook street, Grosvenor square.

1868 HILL, JOHN DANIEL, Surgeon to the Royal Free Hospital; Surgeon to the Royal Orthopædic Hospital; 17, Guilford street, Russell square.

1862 HILL, M. BERKELEY, M.B. Lond., Surgeon to University College Hospital, and Lecturer on Operative Surgery at University College, London; Surgeon for Out-patients to the Lock Hospital; 55, Wimpole street, Cavendish square.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

Elected

1841 †HILTON, JOHN, F,R.S., Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 4.

1859 HIRD, FRANCIS, Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1872 HOGG, FRANCIS ROBERTS, M.D., India.

1843 †HOLDEN, LUTHER, *Vice-President*, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Physician to Casualty Department, St. Bartholomew's Hospital; 32, New Cavendish street, Cavendish square.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road south, Hampstead.

1856 HOLMES, TIMOTHY, *Librarian*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; Professor of Pathology and Surgery to the Royal College of Surgeons; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-4. *Trans.* 5. *Sci. Com.*

1846 HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; 3, George street, Hanover square. C. 1863.

1865 HOWARD, BENJAMIN, M.D., Lecturer on Operative Surgery, and Surgeon to the Long Island College Hospital, New York; 134, West 34th street, New York.

Elected

1865 HOWARD, EDWARD, M.D., Oaklands, Penge, Surrey.

1857 HULKE, JOHN WHITAKER, F.R.S., Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. *Trans.* 4. *Sci. Com.*

1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.

1844 HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 5.

1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.

1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin; 4, Finsbury circus. C. 1870. *Trans.* 5. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., 1, Albert mansions, Victoria street, Pimlico.

Elected

1871 JACKSON, J. HUGHLINGS, M.D., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital; 91, Harley street, Cavendish square.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1839 JEFFREYS, JULIUS, F.R.S., 9, Park villas west, Queen's road, Richmond, Surrey.

1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1864. *Trans.* 3.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."

1847 JOHNSON, GEORGE, M.D., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-4. V.P. 1870. *Trans.* 9.

1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.

1848 JOHNSTONE [JOHNSON], ATHOL ARCHIBALD WOOD, Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.

Elected

1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1837 †JONES, THOMAS WILLIAM, M.D., 55, St. John's park, Upper Holloway. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1872 KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Horsham, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1847 KEYSER, ALFRED, King's Hill, Berkhampstead.

1857 KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.

1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Consulting Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.

1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 18, Harley street, Cavendish square.

Elected

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 95 *bis*, Rue de Passy, Passy, Paris.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Physician to Addenbrooke's Hospital, Cambridge; Deputy for Downing Professor of Medicine, Cambridge University; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Physician-in-Ordinary to H.M. the Queen in Scotland, Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh; 13, Walker street, Edinburgh.

1843 *LEACH, JESSE, Moss Hall, Heywood, Lancashire.

1868 LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.

1843 †LEE, HENRY, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1822 †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Academy of Medicine, Paris; 4, Savile row, Burlington gardens. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1869 LEGG, JOHN WICKHAM, M.D., Physician to Casualty Department and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; 47, Green street, Park lane.

1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

1872 LIEBREICH, RICHARD, Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at St. Thomas's Hospital; 16, Albemarle street, Piccadilly.

1806 LIND, JOHN, M.D.

1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

Elected

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1824 †LOCOCK, SIR CHARLES, Bart., M.D., D.C.L., F.R.S., Member of the Senate of the University of London; 26, Hertford street, Mayfair. C. 1826. V.P. 1841. P. 1857-8. *Trans.* 1.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen, Deputy Inspector-General, and Professor of Clinical and Military Surgery, Army Medical School, Royal Victoria Hospital, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., 4, Graham street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.

1873 MACCARTHY, JEREMIAH, M.A., Assistant-Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.

1867 MACCORMAC, WILLIAM, M.A., Surgeon to, and Joint Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 1.

1846 M'EWEN, WILLIAM, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1866 MACGOWAN, ALEXANDER THORBURN, Kingswood park, near Bristol.

Elected

1823 †MACILWAIN, GEORGE, Consulting Surgeon to the Finsbury Dispensary, and to the St. Anne's Society's Schools; Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN; M.D., Odiham, Hants.

1873 MCKELLAR, ALEXANDER OBERLIN, M.S.I., Resident Assistant-Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

1849 MACLURE, DUNCAN MACLACHLAN, M.B., Lecturer on Physiology at the Westminster Hospital; Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.

1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.

1855 MARCET, WILLIAM, M.D., F.R.S.; 1, Place Massena, Nice. C. 1871. *Trans.* 2. *Sci. Com.*

1867 MARSH, F. HOWARD, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Bruton street, Berkeley square. *Trans.* 1.

1838 MARSH, THOMAS PARR, M.D.

1851 MARSHALL, JOHN, F.R.S., Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; Examiner in Anatomy at the University of London; 10, Savile row, Burlington gardens, C. 1866. *Trans.* 2.

1841 †MARTIN, SIR JAMES RANALD, C.B., F.R.S., Examining Medical Officer to the Secretary of State for India in Council; President of Medical Board for Examination of Officers of H.M.'s Indian Medical Service; Inspector General of Hospitals; 37, Upper Brook street, Grosvenor square. C. 1853. V.P. 1862.

Elected

1864 MASON, FRANCIS, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans.* 1.

1869 MAYO, CHARLES, M.B., New College, Oxford.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent.

1867 MEREDYTH, COLOMIATI, M.D., 76, Margaret street, Cavendish square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868-9. *Trans.* 2.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1873 MILNER, EDWARD, Surgical Registrar, St. Bartholomew's Hospital.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.

1844 MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

1836 MOORE, GEORGE, M.D., Priory Houses, Hastings, Sussex.

1873 MOORE, NORMAN, M.B., St. Bartholomew's Hospital.

1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.

Elected

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.B., Physician to the Manchester Royal Infirmary, and Lecturer on Medicine at the Manchester Royal School of Medicine; 1, St. Peter's square, Manchester.

1874 MORRIS, HENRY, M.A., Senior Assistant-Surgeon to, and Lecturer on Anatomy at, the Middlesex Hospital; 19, Bedford square.

1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans.* 1.

1856 MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital, Consulting Physician to the London Fever Hospital; 79, Wimpole street, Cavendish square. C. 1870-71. *Trans.* 3.

1873 MURRAY, IVOR, M.D., F.R.S. Ed., The Knowle, Brenchley, Kent.

1863 MYERS, ARTHUR B. R., Coldstream Guards' Hospital, Vincent square, Westminster. [Windsor.]

1859 NAYLER, GEORGE, Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 †NEWTON, EDWARD, 4, Upper Wimpole street. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

Elected

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Surgeon to the Brighton Children's Hospital; Surgeon to St. Mary's Hospital, Brighton; 11, Marlborough place, Brighton.

1849 NOVERRE, ARTHUR, 16, Park street, Grosvenor square. C. 1870-71.

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D., 56, Friar Gate, Derby. *Trans.* 2.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Pathology at, St. George's Hospital; Inspector of Anatomy for the Provinces; 30, Cavendish square. C. 1873. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, Derby.

1860 OGLE, WILLIAM, M.D., Lecturer on Physiology at St. George's Hospital; Medical Officer of Health for North Hertfordshire; 25, Gordon street, Gordon square. S. 1868-70. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.B., Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 1.

1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

Elected

1840 †PAGET, SIR JAMES, Bart., D.C.L., F.R.S., Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. *Trans.* 9. *Sci. Com.*

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, House Surgeon to the Hospital for Sick Children; 49, Great Ormond street.

1841 PARKIN, JOHN, M.D., Rome.

1851 PART, JAMES, M.D., 89, Camden road, Camden town.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Physiology at, Guy's Hospital; 35, Grosvenor street.

1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and Lecturer on Materia Medica at, St. Thomas's Hospital; 6, Savile row, Burlington gardens.

1845 PEACOCK, THOMAS BEVILL, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.

1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 18, Temple row, Birmingham. *Trans.* 1.

1870 PERRIN, J. BESWICK, Medical Tutor, Royal School of Medicine; 10, Faulkner street, Manchester.

1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater.

1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]

Elected

1867 PICK, THOMAS PICKERING, Assistant-Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 7, South Eaton place, Eaton square. *Sci. Com.*

1851 *PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.B., Assistant-Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street.

1843 POPE, CHARLES, M.D., The Rectory, East Harptree, Bristol.

1846 POTTER, JEPHSON, M.D., F.L.S., 6, Soho street, Liverpool.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Senior Assistant-Physician to, and Lecturer on Materia Medica at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 15, Henrietta street, Cavendish square.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*

Elected

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874. *Sci. Com.*

1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 47, Kensington park gardens.

1850 QUAIN, RICHARD, M.D., F.R.S., Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

1857 RANKE, HENRY, M.D., Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1869 READ, THOMAS LAURENCE, 11, Petersham terrace, Queen's gate, Kensington.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.

Elected

1869 REEVES, WILLIAM, 5, the Crescent, Carlisle.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 38, Grosvenor street. C. 1870.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., Warcop, Penrith.

1849 *RICHARDSON, WILLIAM, M.D.

1869 RICKARDS, WALTER, M.D., Physician to the Royal Free Hospital; 8, Cavendish place, Cavendish square.

1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1843 †RIDGE, JOSEPH, M.D., 39, Dorset square. C. 1858. *Pro.* 1.

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 1.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1852 ROBERTS, JOHN, M.R.C.P., the Park, Westow hill, Upper Norwood.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

Elected

1843 ROBINSON, GEORGE, M.D. *Trans.* 2.

1843 RODEN, WILLIAM M.D., Morningside, Kidderminster, Worcestershire.

1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Kingston, Surrey.

1850 ROPER, GEORGE, Bank House, Aylsham, Norfolk.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; High street, Hampstead. *Trans.* 1.

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, M.D., F.R.S., Priory House, Cheltenham.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Professor of Medicine at Queen's College, Birmingham; 91, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S.E., Professor of Physiology at King's College, London, and Assistant-Physician to King's College Hospital; Examiner in Physiology at the University of London; Fullerian Professor of Physiology to the Royal Institution; 12, Upper Berkeley street, Portman square.

1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.

1849 SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3.

1855 SANDERSON, JOHN BURDON, M.D., F.R.S., Jodrell Professor of Human Physiology and Histology at University College, London; 49, Queen Anne street, Cavendish square. C. 1869-70. *Trans.* 2. *Sci. Com.* 2.

Elected

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Cheltenham.

1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 29, Duncan terrace, Islington. *Trans.* 1.

1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. *Trans.* 4. *Sci. Com.* 3.

1873 SCOTT, J. M. JOHNSTON, M.D., 13, Eglinton place, Crumlin road, Belfast.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1863 SEDGWICK, WILLIAM, Surgeon to the St. Marylebone Provident Dispensary; 12, Park place, Upper Baker street. *Trans.* 2.

1856 SERCOMBE, EDWIN, 41, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London; Lawnbank, Hampstead. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

Elected

1848 *SHEARMAN, EDWARD JAMES, M.D., F.R.S. Edin., F.L.S., Consulting Physician to the Rotherham Hospital; Moorgate, Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, 12, New Burlington street. *Trans.* 4. *Sci. Com.*

1849 SIBSON, FRANCIS, M.D., F.R.S., *Librarian*, Consulting Physician to St. Mary's Hospital; Member of the Senate of the University of London; 59, Brook street, Grosvenor square. C. 1863-4. L. 1873-4. *Trans.* 1. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., *Vice-President*, Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Trans.* 2. *Sci. Com.*

1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.

1842† SIMON, JOHN, D.C.L., F.R.S., Surgeon to St. Thomas's Hospital; Medical Officer of the Medical Department of the Local Government Board; Whitehall, and 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.

1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.

1872 SMITH, GILBART, M.A., M.B., Visiting Physician to the Margaret Street Infirmary for Consumption; 68, Harley street, Cavendish square.

1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.

1835 SMITH, JOHN GREGORY. [13, Onslow crescent, Brompton.]

1838 †SMITH, SPENCER, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.

Elected

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. *Trans.* 3. *Sci. Com.*

1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.

1845 SMITH, WILLIAM, 70, Pembroke road, Clifton, Bristol. *Trans.* 1.

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, Royal County Hospital, Winchester.

1868 SOLLY, SAMUEL EDWIN, Ivy House, East Sheen.

1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary, and Lecturer on Surgery at the Manchester Royal School of Medicine; 10, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 4.

1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham Common, Surrey. *Trans.* 1.

1843 *SPRANGER, STEPHEN, Cape Town, South Africa.

1867 SQUAREY, CHARLES EDWARD, M.B., 13, Upper Wimpole street. *Trans.* 2

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board. [Greenford House, Sutton, Surrey.]

1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

Elected

1859 STEWART, WILLIAM EDWARD, 12, Weymouth street, Portland place.

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, WILLIAM, Jun., M.D., Professor of Surgery, Royal College of Surgeons, Ireland; Lecturer on Surgery at the Carmichael School of Medicine, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin, *Trans.* 1.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square.

1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.

Elected

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark.

1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripples' Home, Marylebone road; 7, Lower Seymour street, Portman square.

1845 TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square.

1862 THOMPSON, EDMUND SYMES, M.D., *Secretary;* Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 3, Upper George street, Bryanston square. S. 1871-4. *Trans.* 1. *Sci. Com.*

1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 4.

1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 8, Cranley place, Onslow square. *Trans.* 1. *Sci. Com.*

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1872 TOMES, CHARLES S., B.A., Assistant-Surgeon to the Dental Hospital; 37, Cavendish square.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

Elected

1873 TRACY, RICHARD THOMAS, M.D., Lecturer on Obstetrics at the University at Melbourne; Melbourne, Victoria. *Trans.* 1.

1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.

1867 TROTTER, JOHN WILLIAM, Assistant-Surgeon, Coldstream Guards; Hospital, Vincent square, Westminster.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, President of the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.

1873 TURNER, GEORGE BROWN, M.D., Surgeon to the East Sussex Infirmary; 3, Warrior square, St. Leonard's-on-Sea.

1870 VENNING, EDGCOMBE, Assistant-Surgeon, 1st Life Guards; Knightsbridge Barracks, and 24, Belgrave square.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital, and Ophthalmic Surgeon to the West London Hospital; 44A, Wimpole street, Cavendish square.

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

Elected

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.

1867 *WALLIS, GEORGE, Benet street, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy at St. Bartholomew's Hospital; 426, Camden road, Camden town.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

1851 WALTON, HAYNES, Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, M.D. [Agents: Messrs. Grindlay and Co., 55, Parliament street]; 49, Clifton gardens, Maida vale.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square. *Trans.* 1.

Elected

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874. *Trans.* 6.

1835 †WEBSTER, JOHN, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 9, Queen street, St. Andrew's. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-4.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 9. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Physician to the Hospital for Sick Children; 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1828 WHATLEY, JOHN, M.D.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 WILDBORE, FREDERIC, 245, Hackney road.

1870 *WILKIN, JOHN F., M.D. and M.C., The Leas, Folkestone.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.

Elected

1863 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., *President*, Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton; 49, Upper Brook street, Grosvenor square. C. 1849-50. V.P. 1860-1. P. 1873-4. *Trans.* 1. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 78, Park street, Grosvenor square. *Trans.* 2.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square.

1859 WILLIAMS, JOSEPH, M.D. [3, Chichester street, Upper Westbourne terrace.]

1868 WILLIAMS, WILLIAM RHYS, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Bethlehem Royal Hospital, Lambeth road.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Rostellan Castle, Rostellan, County Cork.

Elected

1841 WOOD, GEORGE LEIGHTON, 27, Queen square, Bath.

1851 WOOD, JOHN, F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; Examiner in Anatomy and Physiology at the University of Cambridge; 68, Wimpole street. C. 1867-8. *Trans.* 3.

1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8.

1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, Jun., 62, Bedford gardens, Kensington.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D., Professor of Materia Medica in the University of Edinburgh; Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.

1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.

1868 HOOKER, JOSEPH DALTON, M.D., D.C.L., LL.D., F.R.S., Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 26, Abbey place, St. John's wood.

1868 LYELL, SIR CHARLES, Bart., D.C.L., LL.D., F.R.S., Corresponding Member of the Academies of Sciences of Paris, Berlin, Philadelphia, Boston, &c.; 73, Harley street, Cavendish square.

Elected

1847 OWEN, RICHARD, D.C.L., LL.D., C.B., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1873 AGASSIZ, LOUIS, Professor of Zoology and Geology; Cambridge, Massachusetts. [Deceased.]

1841 ANDRAL, G., M.D., Member of the Institute of France and of the Academy of Medicine; Paris.

1872 BERNARD, CLAUDE, Member of the Institute of France, and of the Academy of Medicine; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Rue de Luxembourg, 24, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1841 EHRENBERG, CHRISTIAN GOTTFRIED, Foreign Associate of the Academy of Sciences of the Institute of France, Berlin.

1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 HELMHOLTZ, H., Professor of Physics and Physiological Optics; Berlin.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

Elected

1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1850 ROKITANSKY, CARL, M.D., Curator of the Imperial Pathological Museum, and late Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.

1856 STROMEYER, LOUIS, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

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CASE

OF

IMPERFECT DEVELOPMENT

OF THE

CIRCULAR MUSCULAR FIBRES OF THE RECTUM AND VAGINA.

BY

HENRY LEE, F.R.C.S.,

SURGEON TO ST. GEORGE'S HOSPITAL.

(Received September 11th—Read October 14th, 1873.)

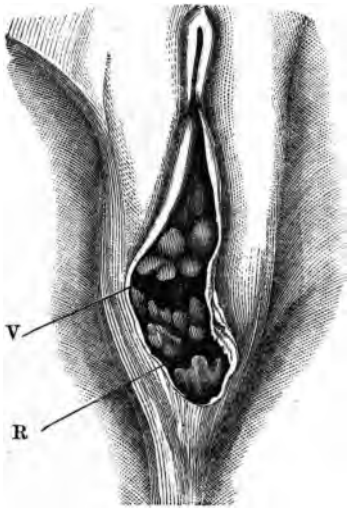
Emma D—, æt. 10, was admitted into St. George's Hospital, on the 9th of August, 1872. There was then a solid movable tumour in the abdomen, extending six inches above the pubis, and five inches broad. This was unaccompanied by any pain or tenderness, and there was no constitutional disturbance.

The mother of the child said that her daughter had not been right from her birth; when six months old nothing passed the bowels for three weeks. Injections and purgatives were then used, and the mother fancied that the part had been lanced (a scoop had probably been used). At twelve months old there was again an obstinate obstruction; since then the patient had been subject to obstruction of the

bowels more or less, but twelve months ago she was worse then she had ever been before. She occasionally had suffered very much pain round the sides, and in the right groin. When admitted into the Hospital she had passed nothing from the bowel for three weeks. She had taken castor-oil and salts repeatedly, sometimes as much as an ounce of either at a time without effect. Upon examination it was found that the sphincters of the rectum and vagina were deficient between the two passages, at the lower part, so that these had a common opening. The accompanying drawing, by Dr. Westmacott (see woodcut), shows the parts as seen from below. The fingers passed with great facility into the rectum, where a large mass of gritty indurated fæces could be felt. This mass was movable, and its motion was communicated to the tumour in the abdomen. It became evident that the abdominal tumour and the distension of the rectum depended upon the same cause, and that the contents of the lower part of the rectum and the bowel above formed one mass.

The indurated fæces in the rectum were broken up, and a large quantity was removed with a scoop. This however produced no sensible effect upon the size of the abdominal tumour. By means of repeated injections and purgatives of different kinds this was however gradually diminished, a large quantity of fæces, very offensive to the smell, being passed daily. On the 24th of August the abdominal tumour had disappeared, and on the 10th of September the patient left the Hospital. On the 12th of October, I heard from her mother that she was again suffering from constipation, accompanied by pain in her side. On the 4th of December this patient was again admitted into St. George's Hospital. The rectum was again distended by a large mass of indurated gritty fæces, and the swelling in the abdomen had reappeared, but not quite to the same extent as upon her first admission. Large doses of castor-oil were administered, and the bowels acted, but the indurated mass of fæces in the rectum remained unaltered. This was now again broken up and removed with a scoop. The gritty nature of the contents of the bowel depended upon the presence of grape

stones and other foreign substances, together with a certain amount of phosphate of lime. After a time the patient was again entirely relieved and left the Hospital, but was again admitted, on the 18th of June, much in the same condition as on the two previous occasions. This time, however, the accumulation was relieved by purgatives and injections, without any mechanical operation. After this had been completely accomplished, on introducing my finger into the rectum and placing my hand above the pubis, I found that they were separated apparently by nothing but the abdominal parietes. This led to a further examination of the vagina and uterus, in which I had the assistance of Dr. J. R. Lee.



The vagina always remained patent. The os uteri could be felt, but above this nothing could be distinguished. It appeared that if the body of the uterus were developed at all it must be in a very rudimentary condition. Examined through the rectum nothing could be felt in the position which the body of the uterus usually occupies. This patient left the Hospital again on the 4th of August.

Instances of a relaxed condition of the bowel, dependent upon deficient or perverted nervous power, the accumulation of fæces, or the repeated use of large injections, are not very uncommon; but the case now recorded is the only one I have met with in which the affection appeared to depend upon an imperfect development of the muscular fibres of the part.

A CASE

OF

IDIOPATHIC HYALITIS

(ACUTE INFLAMMATION OF THE VITREOUS HUMOUR OF BOTH EYES).

BY

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(Received September 17th—Read October 14th, 1873.)

HENRY W—, a powerfully built coal-porter, æt. 33 years, was attacked with dimness of vision on January 22nd, 1873, after a few days of frontal pain, his sight having been previously good.

Personal and family history.—He is married and has six children; has never had syphilis, nor any severe illness, with the exception of small-pox, in an attack of which he was much pitted five years ago. His general health is good. He drinks beer freely, alleging that his severe physical labour renders copious draughts of beer a necessity, but he does not consider himself intemperate. He is in the habit of smoking *about half an ounce of cut-cavendish tobacco a day*. His work is laborious and long-continued, his hours of work being from 5 a.m. till dark, during which time he is constantly lifting and carrying heavy sacks of coals.

Symptoms.—He first noticed "a weakness" of his left eye while reading the paper by candle-light, and left off reading in consequence; but went to work the next morning, though his sight was still somewhat dim. While at work his eyes became inflamed and painful, and his sight so seriously impaired that he was compelled to go home, and suffered severe pain in the forehead for several days before sending for a medical man. Calomel and opium pills were prescribed, but no improvement in his condition took place, partly, as his wife suggested, on account of his disturbed nights, the younger children of the family being very noisy.

State on admission into the South London Ophthalmic Hospital.—On January 31st (nine days after the commencement of the attack), he was led into the hospital, being so nearly blind that he could not safely be left to come even across the room by himself. He had the vacant stare so often observed in amaurotic patients, and on testing his sight he could only read No. 16 of Jaeger's test types with his right eye, and No. 20 only with his left,—the type in each case being held at a distance of about six inches.

Atropine was dropped into both eyes. The right pupil became widely and evenly dilated, but the left was only half dilated, with its margin slightly irregular, as if from swelling of the iris. The tension was normal in each eye. The ophthalmoscope revealed extreme turbidity of the vitreous and crystalline lens in the right eye: no details of the fundus being distinguishable except a faint outline of the optic disc, seen as if through a dense mist. The vitreous of the left eye was also turbid and no details whatever of the fundus could be seen. Slight sclerotic congestion was observable in each eye, but not the vascular zone characteristic of iritis.

Diagnosis.—The suddenness of the attack at first raised a suspicion of glaucoma, but there was a complete absence of glaucomatous tension; diabetes was suspected, but no sugar could be detected in the urine, which had a pale colour and a low specific gravity. There remained then two possible causes of the hyalitis, viz. tobacco-poisoning and syphilis.

I am not aware that tobacco-toxæmia has ever given rise to symptoms and physical changes such as those observed in this case; but it is possible that the cumulative influence of large doses of strong tobacco may have had some share in setting up the local mischief in this instance. Evidences of syphilis were next sought for. The patient steadily denied ever having had any venereal disease whatever, and on examining his genital organs no trace of any venereal sore could be found on them. The only circumstance at all pointing to syphilis was the existence of a scabbed sore, somewhat like that of Rupia, a little below the calf of the right leg; this sore was surrounded by a few smaller pustules, such as are often seen around boils, or any other sore that has been poulticed for some time.

I am therefore still in doubt as to the etiology of the ophthalmic symptoms; though the subsequent history certainly favours the view that syphilis was the principal cause, if not the only one.

Treatment.—Tobacco and alcohol were strictly and absolutely interdicted. Mercurial ointment was rubbed into the skin of the armpits and sides of the chest; and $\frac{1}{16}$ grain of corrosive sublimate in cinnamon-water was given three times daily. Atropine was at the same time applied to the conjunctiva of each eye night and morning.

On the day following the commencement of this treatment, Mr. McHardy, my clinical assistant at the Hospital, made an ophthalmoscopic examination and found that the turbidity of the vitreous had already diminished. The sight had also improved slightly.

In less than a week the sight had almost completely returned, and the gums having become decidedly spongy the mercury was gradually discontinued.

On February 11th (twelve days after the commencement of the treatment in hospital) vision was tested. With either eye the patient could read No. 1 of Jaeger's test types easily. The vitreous and lenses were clear enough to give a distinct view of the details of the fundus in either eye, and the retinæ and choroids appeared healthy. The pupil of the left eye

had become widely dilated and perfectly round, no traces of any adhesions being visible; at the same time the ulcer on the leg had healed and the general health had improved. This improvement continued up to March 4th, when he came to the Hospital as an out-patient. He had already resumed his work, and only complained of being weaker than before the attack.

He was then taking tonics and small doses of iodide of potassium. The tenderness of the gums was going off.

On March 18th his sight was as good as ever, and his general health so much improved that he was ordered to discontinue all medicines.

Remarks.—The rarity of simple inflammation of the vitreous, as evidenced by turbidity, and uncomplicated by choroiditis or iritis, gives this case its chief clinical interest. Not unfrequently in cases of syphilitic iritis there is a slight general turbidity of the transparent media, but in this case the merest trace only of iritis, and no traces whatever of choroiditis, could be detected before the commencement of or after the treatment.

In venturing to call this a case of inflammation of the vitreous I am aware that I am giving a name which, according to some eminent pathologists, has no corresponding disease. Dr. Hermann Pagenstecher (of Wiesbaden) has made some experiments upon rabbits, which seem to him to prove that the vitreous humour is incapable of inflammation in the ordinary acceptation of the term. He injected a drop of croton-oil into the middle of the vitreous, in such a position that he could watch the effects by means of the ophthalmoscope. He found that three days after the introduction of the croton-oil there was no trace of opacity in the immediate neighbourhood of the oil, but the track of the syringe was marked by a line of pus, reaching nearly but not quite up to the position of the foreign body, *i. e.* the drop of croton-oil. At the point of contact of the croton-oil with the vitreous, there was nothing at all like lymphoid corpuscles,—a proof, he maintains, that the vitreous cannot produce pus by its own proper elements, even under the influence of highly irritant

substances. On the other hand, Donders has succeeded in demonstrating a circumscribed suppuration around a foreign body in the vitreous, without any implication of the choroid, so that he concludes that the cellular elements of the vitreous are capable of inflammatory action.

This experiment, however, only proves the possibility of inflammation in the vitreous of traumatic origin; the question whether spontaneously induced inflammation of the vitreous is limited to its proper elements remains unanswered. That there is such a disease as idiopathic hyalitis can only be inferred from cases like that above related. Here we have marked turbidity of the vitreous passing away like a cloud, leaving behind it no traces whatever of any mischief in the choroid, and the sight is completely restored.

Practically, it would make no difference *in the treatment* whether the surrounding tissues were actively inflamed or not, though the prognosis would be very materially influenced; for with acute inflammation of the choroid or retina, or of both, some subsequent impairment of sight must be looked for as inevitable.

It is seldom that an equally good opportunity offers for testing the therapeutical influence of mercury. It raises also an interesting question as to the mode of action of mercury on the non-vascular tissues. If the vitreous were affected in the same way as the gums, *i. e.* by a rapidly increased proliferation of the cells composing it, we should expect that the turbidity would be rather increased than diminished. Hence we must conclude that, (1) mercury affects the vascular and non-vascular tissues in an entirely different way; or (2) that the non-vascular tissues are only directly influenced by mercury through the medium of the nearest blood-vessels. I am inclined to the latter view, and beg to suggest that under the influence of this medicine the choroidal vessels and absorbents have a greatly increased absorbing power, and that the effused products in their neighbourhood are thus rapidly removed.

That effused products often disappear under the influence

of mercury is a matter of daily experience, but it is not often that the result is obtained so rapidly and so completely as in the present instance.

NOTES OF A CASE

OF

DUCHENNE'S PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS,

WITH SPECIAL REFERENCE TO THE TEMPERATURE OF THE OVERGROWN LIMBS, AND WITH GENERAL REMARKS.

BY

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(Received October 13th—Read October 28th, 1873.)

SINCE Dr. Duchenne first drew attention, in the year 1861, to the occurrence of a form of progressive paralysis, accompanied by hypertrophy of certain muscles, several English observers have brought before the notice of the profession illustrative cases. Among these I may mention Dr. Meryon, Mr. Wm. Adams, Dr. Hillier, Dr. Langdon Down, Mr. Kesteven, and Mr. Butlin. But the pathology of the disease is only in part understood, and the disease itself is of sufficient rarity to justify me in communicating to this Society the record of a case in which one new, or at least seldom noticed, fact has been observed.

A. M—, æt. 7, was admitted for the second time into St. Thomas's Hospital, under Dr. Bristowe, on the 1st of May, 1873.

History.—His mother stated that he was a "beautiful baby" when born; that he began to say words at nine months, and "spoke properly" before he was two years old. He had no fits during infancy or afterwards. About two years ago, a year after an attack of measles, he began to show signs of weakness in his limbs. To use the mother's very apt words—"his right foot hanged with him." He soon began to tumble down while walking, but was at first able to get up again without help. About ten months ago it was noticed that he could no longer pick himself up again after a fall, and about the same time the calves of both legs were seen to be over-grown (See Plate I). No particular extension of weakness since then appears to have been recognised by his parents. His mother says that he learned the alphabet at six years of age, and she considers that he has a good memory.

He has two brothers living: one, aged ten years, an idiot from early childhood; the other, aged eight months, as yet a "beautiful baby." One sister is living, aged three years, healthy. One sister and one brother died in infancy, one of whooping-cough, the other of teething. The mother knows of nothing which could have caused his illness. He was in Victoria Ward from November 25th, 1872, to January 12th, 1873, and is said by the sister of the ward to be stronger now than then.

Examination.—The boy stands with his feet placed about nine inches apart, the toes being turned a little out; the feet are large and flat. The knees are bent a little forwards, the buttocks backwards; the calves, very much overgrown as compared with all other muscular parts of the body, project far beyond the line of the thighs in profile. His back forms a deep curve forward from the hip to the shoulders, so that the belly and lower chest are made very prominent, and the shoulders are thrown back. A line falling from the point of the shoulder to the ground would be quite clear of the buttock. The head, rather large in proportion to the body, is bent forwards. The muscles of the back are evidently much wasted.

The right shoulder is contracted and drawn downwards, and its muscles are also wasted. The muscles of the rest of the body, excepting the calves, are poor and thin for a boy of his age (See Plate I).

Measurements :—

Circumference of head from occipital protuberance round frontal protuberances . . .	20 inches.
Right upper arm below insertion of pectoralis major . .	$5\frac{1}{8}$,,
Left ditto	$5\frac{1}{2}$,,
Right forearm two inches below elbow	$5\frac{1}{4}$,,
Left forearm two inches below elbow	$5\frac{1}{4}$,,
Circumference of chest, nipple line	$20\frac{1}{4}$,,
Circumference of abdomen at umbilicus	$17\frac{3}{4}$,,
Right thigh, middle . . .	$9\frac{1}{4}$,,
Left thigh, middle . . .	$9\frac{1}{2}$,,
Right leg	$8\frac{3}{4}$,,
Left leg	$9\frac{1}{16}$,,[1]

He walks from his hips, shuffling the feet along the ground without getting them clear, and swaying his body from side to side in the alternate raising and lowering of the hips. There is evident weakness in the movements of all muscles below the

[1] I have since measured the legs and thighs of a number of healthy children, and find that the calf is usually about a fourth less in girth than the thigh, the ratio being in six cases 12 to $8\frac{3}{4}$. Comparing slim children of about the same age, I found that the calves in this case were at least half an inch larger than they should be. They appeared to be overgrown therefore—1st, as compared with the rest of the muscles of the body; and 2nd, as compared with their natural bulk. They were much firmer to the touch than the other muscles of the body, and when thrown into contraction became very hard and knotty.

hips. The toes are turned inwards in walking. On the lightest touch, and often without external impulse, he falls down. The left leg is the first part to give way; it is doubled under him, while the right is extended from the knee and flexed from the hip; at the same time the body comes crashing to the ground in a sitting posture. In this his favorite attitude he moves readily along the floor by the aid of his arms. When he wishes to rise he bends his trunk forwards, and places his hands upon the floor in a way that reminds me of a baboon; he then bends the right leg and raises the trunk in an oblique position, but cannot rise to his feet; then, by seizing a chair or similar support with both hands, he is enabled to straighten his legs and stand, but with the trunk now horizontal. The sacro-spinal muscles are too weak to lift the head and shoulders without further assistance. It is necessary therefore, lastly, that he seize some higher fulcrum (such as a bedpost), and climbing, with hand over hand, gradually raise the shoulders through the agency of the dorso-humeral muscles.

As concerns his intellect few indications of deficiency or damage can be detected. His manner is gentle and somewhat lively; he is well-conducted and obedient, but the sister of the ward says that his memory is short and that he stammers at times. His speech is clear and free from stuttering.

All the muscles of the body are sensible to faradic electricity. The muscles of the calf are the most sensitive; the left thigh more than the right; the right pectoralis major, deltoid, and sacro-spinal muscles, less than the rest. The left leg and the right arm are distinctly weaker than their fellows. When the sensibility of the legs and other parts is tested with callipers his answers are so vague and contradictory as to make it quite uncertain whether there be any paralysis or excess of sensibility.

Observations.—The skin of the calves was not different in tint from the skin elsewhere, that is to say there were no signs of the red discoloration spoken of by Duchenne. On

the other hand, when the hand was applied to them they appeared to be warmer than the thighs. I had not been able to find any observations of the existence of a higher temperature in the skin over the hypertrophied muscles, either in Duchenne's works or others, although the possibility of the occurrence had not escaped that acute observer. I now endeavoured to ascertain by the use of a thermometer the relative temperatures of the legs and thighs. For this purpose I made use of a surface thermometer, made by Mr. Hawksley, principally, I believe, at the suggestion of Dr. Sibson. It consists of an ordinary clinical thermometer having the bulb lengthened and twisted into a flat coil. One side of the coil is supported by a little plate of boxwood, to which it is cemented by putty. Over the wooden plate an oval sheet of india-rubber is stretched, having its margin extended by a ring of metal fixed to the stem of the thermometer. When the thermometer is applied with the free face of the coil in contact with the skin, the coil is enclosed by the above arrangement in a little chamber with non-conducting walls, and is defended from atmospheric and other disturbing influences. In taking the temperature the thermometer was always brought to 80° Fahr. as a starting-point, and the further rise of temperature during three minutes' application of the instrument to the surface was noted.

The first observation was made on the 3rd of September, when the thighs and calves were compared. The results were—

Right thigh 83·0	Left thigh 83·6
Right calf 85·6	Left calf 85·7

showing a difference in favour of the calf of 2·1, and 2·6, on the left and right sides respectively.

On September 8th, right thigh 85·3; right calf 87·1; a difference of 1·8.

The observation was repeated at the end of ten minutes, during which the child being under examination was exposed in a temperature of 62° Fahr.

The figures were now—

1st. Right thigh 81·5, after 3 minutes of exposure.
2nd. „ 82·5, after 3¾ „ „
3rd. Right calf 85·4, after 3 „ „

Both parts being cooler, the difference was more marked than before, amounting to nearly four degrees with equal exposure.

September 11th.—Left thigh 87·5; left calf 89·7; 2·2 deg.
September 13th.—Left thigh 86·3 (3¼ minutes).
Left calf 89·2 (3 „). (2·9 deg.
Left shoulder 87·6
Left cheek 92·5, after a fit of crying, the face being flushed.

Similar observations were made on three other occasions, with the same result. There was an excess of temperature in the calf of from 1·8 to 3·9, as compared with the thigh.

The microscopical condition of the muscles of the calf was next investigated. For this purpose an emporte-piece, or tissue-explorer, devised by Mr. Hawksley, was used, Dr. Duchenne's instrument appearing to be unnecessarily large. Mr. Hawksley has made for me a small cannula, with oblique-pointed and sharp-edged end, through which, when thrust into the tissues, a stylet, terminating in a screw with a broad sharpened thread, might be introduced, and by a twisting movement might be made to entangle small pieces. In practice this worked very well. It gave little pain, caused little or no bleeding, and after bringing away enough tissue to suffice for three or more microscopic slides, it left only a tiny puncture, which healed without any discomfort.

The muscular tissue brought away was paler than natural. Under the microscope the muscular fibres were of full size, well defined, and free from fat or other accompaniments of degeneration. The striæ were fine, clear, and close. Between the fibres an unusual proportion of fibrous tissue was observed, and it appeared also to me that there was an excess of nuclei on the surface of the sarcolemmata. There did not appear to be an excess of fat.

I have spoken of this case as illustrating Dr. Duchenne's pseudo-hypertrophic muscular paralysis, because the symptoms agree with those of the cases published by him. The disease with which this disease might be most easily confounded, would be the progressive muscular atrophy of infancy. But, as Duchenne shows, this disease advances from the face to the trunk, from the upper limbs to the lower; it destroys the muscles in a progressive but irregular way, and, after the destruction of the muscular fibres, produces local and partial deformities. Whereas, in the case under consideration, as in other similar cases, the disease attacks first and principally the lower limbs, producing a gradual and general enfeeblement, and an overgrowth instead of a wasting of certain muscles. The atrophic paralysis of infancy (infantile paralysis) is even more clearly defined by its sudden occurrence—fits or fever often introducing it; by its completeness at first, tending to diminution afterwards, with localisation in a certain number of muscles; this localisation issuing at a later stage in various partial deformities of limbs, owing to disturbance of the balance of muscular tonic force. In this I again quote Duchenne. The considerations which led me to read the note of this case before the Society were connected with the observations of the temperature of the calves. The disease is sufficiently rare to furnish individual observers with but few illustrations in a long time. The pathology of the disease cannot be said as yet to be understood. And therefore any observations tending to throw light upon this point will be useful if they are followed up by those who have at one time or another opportunities of investigation. It is cited by Duchenne as one of the points of difference between this disease and progressive muscular atrophy, that whereas in the latter the papers of Dr. Lockhart Clarke and Dr. Bastian have proved the existence of degeneration and wasting of the anterior column and grey matter of the cord, in examination of the spinal cord of subjects of the pseudo-hypertrophic muscular paralysis made under Dr. Duchenne's own directions, no morbid appearances have been detected. In one of the latest reported, only a year ago, the sections of the spinal

cord are recorded to have been altogether healthy, and the testimony of Cohnheim and Eulenburg is to the same effect. Mr. Kesteven, a most trustworthy authority, reported in the 'Journal of Mental Science' for January, 1871, upon th "Microscopical Anatomy of the Brain and Spinal Cord, in a Case of Duchenne's Paralysis, associated with Imbecility." He found the following morbid conditions and changes of structure:—dilatation of the peri-vascular canals, consequent upon long-standing congestion; and numerous circumscribed spots of granular degeneration of the nerve substance. These were found irregularly scattered in the white substance adjoining the grey matter of the convolutions, while very few were noticed in the grey matter itself. They were sparse in the corpus striatum and thalamus opticus; and in the medulla and cord they were few and far between. They were evidently spaces caused by loss of tissue replaced by the morbid matter. It must be remarked that Mr. Kesteven found similar appearances in a case of idiocy with convulsions, and in other cases, so that it cannot be deemed to be characteristic of the pseudo-hypertrophic paralysis.

Of the condition of the muscles in this disease we have evidence from many observers. From a comparison of various statements it results that the appearance differs in the several stages of the disease. There is an early stage in which the muscular tissue is little altered, but there is excess of intermuscular fibrous tissue; and there are further stages in which the muscular tissue becomes attenuated and degenerated, and in which there is present a growing quantity of fat between the fibres. In advanced stages, described by Cohnheim, there is actually a large quantity of adipose tissue formed between the muscular fibres. This latter form would appear almost a distinct one. Between the irregular deposition of fat in granules and the formation of adipose tissue the difference is of course between degeneration and tissue-formation; the formation of fat-cells from the corpuscles of the connective tissue being a very generally accepted fact. The present case, however, does not throw light upon this point. The history and the histology both demonstrate an

early stage of the disease in which the muscular fibres are still perfect, and the overgrowth of fibre simple and untainted by intrusion of fat.

With this I am inclined to connect the comparatively high temperature of the calves. It would indicate, in one way, what the "hyperplasia," to use Duchenne's word, indicates in another, hyperæmia of the affected muscles. In relation to this I noted very carefully the power of the muscles. Under the influence of the will and under the stimulation of electricity they contracted firmly, and, as I have noted, became knotty; but the power they exerted was but feeble. More than once I have heard it argued that the hypertrophy was a real hypertrophy, and was due to the extra work thrown upon certain muscles by the weakness of others. The cases of general pseudo-hypertrophy described by Duchenne made it certain that at least in one group of cases this would not be the explanation. And after watching for a long time on many occasions the movements of the child, I arrived at a certain conclusion that the calves were, relatively, not stronger than the other muscles, and that they did not do more duty. He made use to an overwhelming extent of the thin but much stronger muscles of the upper limbs. After this I think that the conclusion is fair that first the hyperæmia, and second, the overgrowth are due to internal causes —to causes affecting nervous centres. And I would further suggest that it is possible that the disease may have its origin in the sympathetic nervous system, outside the spinal cord and brain. The whole story is rather one of weakness following faulty nutrition, than of paralysis of muscular nerves, followed by wasting of muscles. There is at least evidence of vasomotor disturbance in connection with the overgrown calves, and I should consider any future post-mortem examination incomplete which did not include a careful investigation of the state of the prævertebral and other ganglia. Such an examination was in one case made by Cohnheim, who reported that he had not found any alteration of the ganglionic system.

In the present case, the possibility of vasomotor distur-

bance being set up by peripheric irritation was not lost sight of. None could be detected, however. The child's viscera gave all the indications of health, and excepting locomotion, the functions were well performed.

In referring the disease mainly to vasomotor disturbance I am but following the indications of Dr. Duchenne, whose views appear to me to be confirmed by the observations in this case.

DESCRIPTION OF PLATE I.

Case of Duchenne's Pseudo-Hypertrophic Muscular Paralysis.

Fig. 1. Front view.
Fig. 2. Back view.
Fig. 3. Side view.

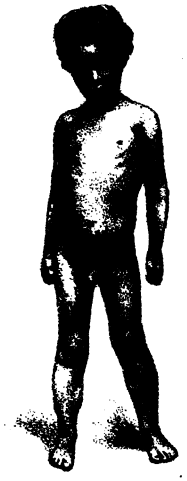

Fig. 1.

Fig. 2.

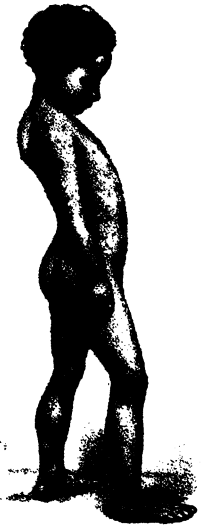

Fig. 3.

G.H Ford.

Mintern Bros imp.

ON THE

ACOUSTIC PRINCIPLES AND CONSTRUCTION

OF

STETHOSCOPES AND EAR TRUMPETS.

BY

CHARLES J. B. WILLIAMS, M.D., F.R.S.,

PRESIDENT OF THE SOCIETY.

(Received October 21st—Read October 28th, 1875.)

THE stethoscope, or chest explorer, is the well-known acoustic instrument invented by Laennec, to convey sounds from the chest of the patient to the ear of the observer. After his discovery of the utility of such an instrument by using a thick roll of paper, he substituted a cylinder of wood, perforated lengthways, and excavated at its pectoral end. At first he made this hollow conical, but afterwards preferred a parabolic form, as more easily made, and not inferior in efficacy.

The first great improvement in the stethoscope was that of Piorry, which reduced its bulk from a solid cylinder to a slender stem, with an expansion at each end for the ear and chest; and such an instrument, variously modified, has been in common use ever since.

After a vast number of experiments and comparisons of

instruments of different forms, about thirty years ago I devised the trumpet shape for the pectoral end;[1] and I have found reasons ever since to prefer it, on account both of its greater efficacy, and of its facility of application. A series of investigations convinced me that the stethoscope conducts the sounds from the chest both through its solid fibres, and through its column of contained air—some sounds passing best by one medium, and some by the other. Assuming for the present this to be the principle of its operation, it may be useful to consider how these objects may be best combined in the same instrument; and first, with regard to the *material.*

As a conductor of sound, wood has been proved to be superior to all solids, except iron. Fir, aspen, cedar, and such kinds of wood as are rigid in longitudinal fibre, and at the same time light in weight, are the best, for the same reason that renders them the proper material for the sounding-boards of musical instruments; they readily receive and transmit sonorous vibrations from other bodies, without any modifying note or ring of their own, such as we find in metals and glass. But the great objection to wood, is its fragility; especially when reduced to the shape best suited for the purposes of the stethoscope. Metals are also good conductors; but besides their tinkling note just noticed, their weight, and their coldness when first applied to the chest and ear, are objectionable. Ivory, horn, gutta-percha, or papier-maché, would answer well; but the most suitable material of all seems to be the preparation of india-rubber, called *ebonite,* being easily formed into any shape; and being also light, durable, and a good conductor of sound.

We next have to consider the *form* of the instrument. It is obviously important that a stethoscope, which is to convey sounds from the chest to the ear, should be of such a form as to ensure complete contact with both chest and ear; and this not only because the conduction of sound is thereby facilitated through the solids, but also because the enclosed

[1] 'Med. Gazette,' 1842, p. 400.

column of air is thus made *air-tight*, and excluding all external noises and motions, is the more sensitive to the vibrations proceeding from within the chest. Every auscultator knows how imperfect contact from tilting, or from inequalities of the chest in thin persons, almost destroys the conducting power of the stethoscope, and gives entrance to confusing external noises.[1] The trumpet-shaped pectoral end secures this perfect contact by bringing both the solid and the contained air of the instrument into the closest apposition to the walls of the chest. This form is also more comfortable to the patient, by its flatter contact, than that of the conical stethoscopes, the edges of which often give pain. For the same reason its application is easier and more steady for the observer, who may even augment the intensity of the sound, when desired, by increased pressure, without inconvenience.

The power of an enclosed column of air to transmit sound to considerable distances, notwithstanding curves and angles in its course, as shown in flexible ear-tubes and speaking-pipes, proves that the conveyance of sound through tubes is not merely in straight lines undergoing reflection, as in open air, as was formerly supposed, but takes place in waves, through the whole body of the confined air, vibrating through curves and angles with very little loss of force; so that through many feet of tube a whisper may be heard which would be inaudible at half the distance in the open air. But the loudness of the sounds so transmitted, is not accompanied by an equal degree of distinctness. On the

[1] Many years ago, I fitted to the pectoral end of a stethoscope a diaphragm of thin india-rubber, which, by enclosing the air, enabled it to transmit sounds from the chest without complete contact; and I found this instrument peculiarly effective in examining very thin persons, or others, through garments. The same result may be obtained, with some intensification of the sounds, by filling the end of the stethoscope with a small hollow sphere of the thinnest india-rubber. This closes the column of air by an elastic drum, highly sensitive to the vibrations of sound. I understand that M. König, of Paris, has constructed a stethoscope with an india-rubber diaphragm on the same principle.

contrary, flexible tubes, and others also when long or with a large hollow, have always more or less reverberation, a note or echo of their own, which confuses and obscures the original sounds. I shall presently have to notice this as the great fault of ordinary ear trumpets; but it exists also to some extent in stethoscopes with long tubes or large hollows.

In the small trumpet-shaped stethoscope we have the best kind and form of solid, combined with the smallest volume of enclosed air, fitting it to receive and transmit the sounds from the chest, directly, simply, and without alteration. I have been using it, constructed in wood, during the last thirty years; my only cause of dissatisfaction having been that it has so frequently got broken. For the sake of greater durability and elegance, I now recommend *ebonite*, which I have found to answer equally well.

It is constructed in two forms. One is a single piece, which may be carried in the hat. The other is made more portable for the pocket by the ear-piece taking off (somewhat conical like the stopper of a bottle without screw), and slipping into the hollow trumpet end; this secures it from injury in case of its falling or being sat upon. In this form, applied with its ear end to the chest, it may be used to indicate the direction and limits of the pulsations of the heart or of aneurisms.

In ordinary use the trumpet end concentrates, and thereby somewhat augments the diffused sounds of the chest, such as the breath sound. By reversing the tube, and applying the aural end to the chest, we have the means of avoiding this concentration, and of isolating the sound of a small spot, as Laennec used to do by adding a perforated stopper to his cylinder. This especially is sometimes useful in distinguishing the sounds of cavities and of valvular murmurs. But the same object may be better attained by using the stethoscope with the earpiece in the pectoral end, as carried in the pocket; for the small end has also a little trumpet expansion, adapted to explore small spots or hollows on the chest.

FIG. 1. FIG. 2.

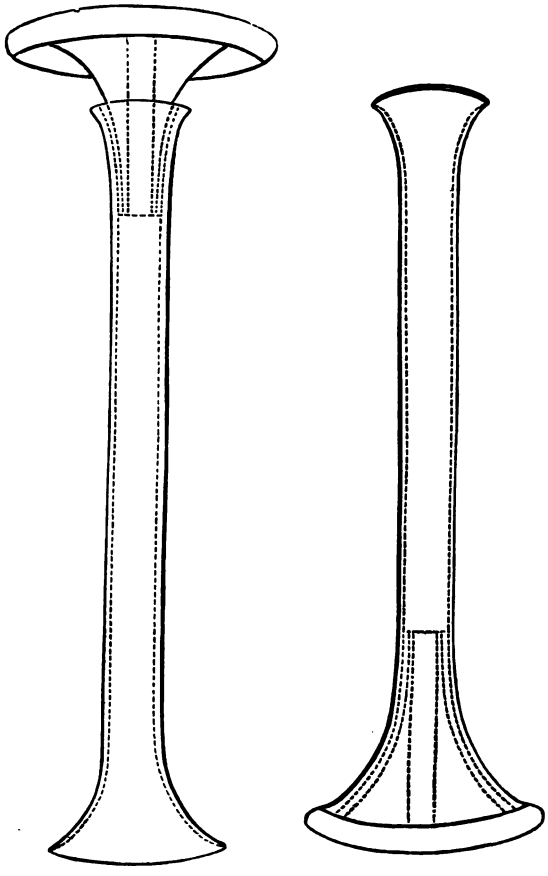

FIG. 1.—Stethoscope as commonly used.

FIG. 2.—The stethoscope packed for the pocket. In this form it is also used for exploring small spots with its small end; or if applied by its broad end, it may be used to indicate the pulsations of the heart or of aneurisms.

EAR TRUMPETS.

The acoustic principle of the ear trumpet is quite distinct from that of the stethoscope. Instead of being both a solid conductor and a closed tube of air, like the stethoscope, it is an aerial reflector and an open tube, to catch the waves of sound in the open air, and to concentrate them with increased force into the ear. Sounds are ordinarily conveyed to the ear through the air, and as this is both a slower and a weaker conductor of sound than solids, we have but few means of intensifying its sounds, and the chief of these is by reflection and concentration.

The best *materials* for reflecting aerial sounds, are those solids which most differ from air in hardness and density, such as metals, glass, porcelain, &c.; but the weight of some of these, the fragility of others, and the intrinsic tinkling note of all, form objections to their use as ear trumpets.

Still, the best instruments in common use are made of silver, plated metal, or japanned iron. The lightness of aluminium might claim for it a preference. Polished ebonite also forms a good reflecting surface; and although inferior in power to metal, it has advantages over it in point of lightness, and freedom from intrinsic sounds.

The *form* of the ear trumpet requires careful consideration. The simplest and most efficient reflector is a hollow cone, with a wide base open to receive as large a body of sound as possible, which is reflected directly to the apex, and conveyed through a short curved tube to the ear. All repeated or secondary reflections of sound, such as those in parabolic or spheroidal cavities, are to be avoided; because being retarded, they confuse the sound by an echo following, instead of adding to its distinctness. Such instruments may increase the noise, but they impair the clearness of the articulate sounds, which hearing trumpets ought to convey to the ear pure, and unmixed with echoes or extraneous noises.

Now, in point of fact, these confusing sound are heard, more or less, in all ear trumpets, as commonly constructed.

They all have the *conch-like roar*, resembling that of the sea heard in large shells, which confounds the clearness of words or other definite sounds. This noise is a reverberating echo of any sounds from without, and takes its tone from the note proper to the size of the cavity or tube. Every tube has its proper note, resulting from the vibrations of its columns of air from end to end, and every cavity has its note produced by repeated reflections across its diameter; and these notes respond to every noise or impulse communicated to the tube or cavity. Now, these noises not only confuse the original sound, but are often painful to the ear, which may be sensitive in feeling, although its hearing is impaired. It is therefore an important object to get rid of them, and I have now to bring before you various means, which I have devised to remove these confusing sounds from my conical ear tube. I may premise that one circumstance favoring this result, may be found in the shape of the cone, which is large, subtending an angle of from twenty to twenty-five degrees. When narrower, the cone takes in less sound, and has more internal reverberation. But even in wide cones, there remains much of this noise that needs correction, and the following expedients have been tried with this object.

1. Perforating the sides of the cone with a few scattered holes to give vent and diversion to the transverse vibrations.

2. By introducing a diaphragm of stiff paper longitudinally, in the axis of the cone.

3. By using a less perfect reflector, such as stout paper, for the expansion of the cone at its broad end.[1]

4. By opening one side of the cone by oblique truncation, and prolonging the opening by a slit to within a few inches of the ear.

Each of these experiments succeeds more or less in removing the confusing sound; but they all also impair the power of the instrument as a reflector. For practical

[1] I exhibited ear trumpets thus corrected at the Meeting of the British Association for the Advancement of Science, at Bristol, in 1836.

purposes the last plan answers best—leaving open a considerable length of one side of the cone. In so doing, we get an instrument which has an obvious resemblance to the ear of many quadrupeds; and there can be little doubt that this form of external ear is wisely designed to aid their hearing in the simplest and most efficient way, by directing and concentrating sounds, without the confusing reverberation produced in complete tubes and cavities. It is not improbable, too, that the hairs which fringe the margins of the ears in some animals, may subserve a like purpose, in muffling stray and superfluous sounds. If found necessary, we might profit by this hint, by trimming our ear tubes with a silk fringe or some similar drapery.

I have made some attempts to estimate the magnifying power of these instruments; but it is difficult to make accurate observations in a city where disturbing noises are rarely absent. Taking as a test sound the ticking of a small French clock, by the aid of these tubes I have heard it at from ten to a hundred times[1] the distance at which it could be heard by the unaided ear. A large pasteboard cone, twenty-two inches long, and ten inches broad at its base, made the sound audible at about a hundred times the distance at which the unassisted ear could hear it; whilst the magnifying power of smaller ebonite ear trumpets for use, measuring respectively twelve inches by four, and nine by three, showed magnifying powers of twenty and ten times—which are sufficient to enable most deaf persons to hear more or less distinctly.

In the case of all aids to hearing hitherto noticed, the use of the external ear, or pinna, is superseded by the small tube of the ear trumpet being inserted into the auditory passage. But we ought not altogether to disregard the aid to our hearing which nature has provided in our outer ears, although she has been less bountiful to man than to many of the lower animals. Judging from my own experience and that of others whose hearing is impaired, I think that much

[1] These numbers are much higher than those first given, and are the result of more careful experiments.

help might be obtained from the use of a sound collector behind the ear, and directing it forward in the direction of the sound, as we instinctively do with the hand with good effect. A light cowl of india rubber or gutta percha, of semi-parabolic form, with a rounded notch to fit behind the ear, and with its free margin projecting three or four inches in a parabolic curve beyond the pinna, would probably prove a considerable aid to the hearing; but the ear, although directed forward, must be freely open in front.

I feel that an apology is due to the Society for the hasty manner in which these observations have been put together. They were not hastily made, but are the result of many years' experience and thought; and although an imperfect endeavour to clear up a confessedly obscure subject, I hope its discussion may induce others to cultivate the application of acoustics to medicine with further success.

(At the conclusion of the paper Dr. Williams exhibited and explained various forms of stethoscopes and ear trumpets, an account of which will be found in the 'Proceedings,' vol vii, p. 189.)

N.B.—The ebonite stethoscopes and ear trumpets above described are made by the India Rubber, Gutta Percha, and Telegraph Works Company, Silvertown, and 100, Cannon Street, E.C.

Fig. 3.

Fig. 4.

Figs. 3 and 4.—Outline of Ear Trumpets, half the real size of the larger and two thirds that of the smaller one.

A SUCCESSFUL CASE

OF

ABDOMINAL SECTION FOR INTUSSUSCEPTION;

WITH REMARKS ON THIS AND OTHER METHODS OF TREATMENT.

BY

JONATHAN HUTCHINSON, F.R.C.S.,

SENIOR SURGEON TO THE LONDON HOSPITAL; SURGEON TO MOORFIELDS OPHTHALMIC HOSPITAL AND TO THE HOSPITAL FOR SKIN DISEASES.

Received October 21st—Read November 11th, 1873.

THE case of intussusception which I am about to describe came under my care at the London Hospital in 1871.

The patient was a somewhat delicate female child aged two years. She had previously been seen by my colleague Mr. Waren Tay, who had diagnosed her disease, and by whom she was transferred to my care in order that she might be admitted as an in-patient.

From her anus there protruded a portion of bowel about two inches long, deeply congested and much swollen. By the side of this the finger could be passed, its full length, into the rectum without reaching the point at which the intussusception began. On carefully examining the extremity of the protruded part, I noticed that it did not present merely a rounded opening as usual in such cases. I was able easily to identify the pouch and valve of the cæcum, with the

opening into the ileum. Of these parts it was of course the mucous membrane which was visible, and the appendix cœci was wholly concealed between the folds of the intussusception. This discovery rendered it evident that we had to deal with an involution of bowel of very unusual length, which commencing at the cæcum had allowed the ileum to pass through the entire length of the colon, and actually to become extruded at the anus.

On examination of the child's abdomen externally the tract of bowel involved could be felt like a long firm sausage passing down the left side.

The mother of the child gave us the history that the latter had begun to suffer from pains in the abdomen, rather suddenly, about a month previously. Her first attack of pain was one Sunday afternoon, and was such as to cause screaming. It was quickly followed by a motion which contained blood, and by frequent vomiting. A fortnight after this, the child having been ailing the whole time, a protrusion of bowel was noticed at the anus. This was reduced by the surgeon then in attendance, and a cork pad was fitted over it. It was found impossible, however, to prevent the prolapse from recurring, and the child continued to be sick and to pass blood-stained mucus.

Three days before admission the prolapse increased to such a size that the parents were unable to reduce it, and were obliged on three occasions to call in surgical aid for that purpose. There had been no real obstruction of the bowels, but only temporary constipation at times.

The child, at the time of her admission, looked very ill. Her countenance was pale and anxious, and from her mother's description it was evident that her strength had been failing rapidly during the last few days. It appeared that she was almost constantly engaged in straining to get rid of the bowel which filled the rectum.

Our first measure of treatment consisted in putting the child under chloroform, and then, whilst she was held up by the feet, distending the rectum to the utmost with warm water.

WATSON, JAMES, 1845-
The dog book; a popular history of the dog, with practical information as to care and management of house, kennel, and exhibition dogs; and descriptions of all the important breeds... London, Heinemann, 1906.
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of the bowel involved. The opposed serous surfaces did not present a single flake of lymph, and they were congested in only a moderate degree.

Having completed the reduction, I put the bowel back into the abdomen, and closed the wound with harelip pins and interrupted sutures.

The operation had been an extremely simple one, and had not occupied more than two or three minutes.

The abdomen having been well supported by strapping,

cotton wool, and a flannel bandage, the child was returned to bed.

The after-treatment consisted in the use of milk enemata every three hours, with the occasional addition of five minims of tincture of opium.

No vomiting occurred after the operation. No food whatever was allowed to be taken by the mouth during the next two days. The temperature, on the evening of the operation, was 100·5°, but subsequently fell to 99°, and with the exception of the fifth day, on the evening of which it rose to 101·7°, it never exceeded 100°. Chloroform was administered on two or three occasions to allow of the wound being dressed without the child's screaming. The pins were taken out on the fourth day, that is, seventy-two hours after the operation.

I had felt much anxiety as to the healing of the abdominal wound on account of the thinness of the parietes, but nothing untoward occurred.

The child recovered without having ever showed the slightest symptom of peritonitis, and left the hospital in excellent health about three weeks after the operation.

Nothing but fluid food (milk and beef tea) had been allowed through the greater part of this time from a fear of producing any return of the intussusception. The child was fitted with an abdominal support when discharged, but the scar was sound and strong, and there was no tendency to bulging.

The successful issue of a single case goes but a very little way towards proof that the line of practice adopted was the proper one. I purpose, therefore, with the permission of the Society, to discuss this question in some detail; and the first items of evidence which I will mention are the cases which had previously come under my own observation.

About twelve years ago I operated for harelip upon a delicate child about ten months old. The child had been brought from Somersetshire on purpose to have the operation done, otherwise its feeble state of health would have caused

me to defer it. The lip healed well, but about the tenth day the child began to pass blood and slime. On examination *per anum* I found a long intussusception occupying the rectum. It never protruded at the anus. I tried during six days a great variety of means with a view to the replacement of the involution. Chloroform was repeatedly given. Injections of air and of water were made over and over again, and in various positions of the body, and attempts were also made with a long tube to push the bowel into place. Several times I thought I had succeeded, and on one occasion the passage of a considérable quantity of fæces made me feel confident that reduction had been effected. About six hours after this occurred, however, the child, who had been gradually failing in strength, died. No post-mortem was permitted, and I am unable to state whether the reduction was complete. My colleague, Mr. John Adams, had, on one occasion, seen the patient in consultation with me, and had assisted in attempts at reduction.

A few years later I saw another similar case in consultation with Dr. James and Dr. Bright, at Forest Hill. Our patient was a remarkably fine healthy boy, of about three years of age. A long intussusception occupied his rectum, and came low down, although it never actually protruded. Again we tried every plan that we could think of, but without success. Enemata were used in the most forcible manner with the child's body inverted, and they were repeated several times a day, and often under the influence of chloroform. Bougies of various kinds were also carefully tried.

The child sank from exhaustion about a fortnight after the commencement of the symptoms.

Dr. James obtained a post-mortem which confirmed the diagnosis as to the condition of parts.

A third case was brought under my notice by Mr. Waren Tay three years ago. Its subject was a female child aged about fifteen months. Blood had been noticed escaping from the bowel on the day before Mr. Tay was consulted. The child had great pain and was repeatedly sick. On

examination of the bowel, Mr. Tay discovered at the distance of two or three inches from the anus the extremity of a long intussusception. The portion of bowel involved could also be easily felt through the abdominal wall. Repeated attempts were made by manipulation and by injections to effect replacement of the bowel, but without success, and on the seventh day from the commencement of the symptoms the child died. The autopsy showed an intussusception of the transverse into the descending colon, involving, however, only about two inches of the bowel. It was thought probable that the greater portion had been reduced. The small intestines were much distended, and were somewhat congested on their peritoneal surface, but were quite free from lymph. There were no adhesions whatever of the opposed peritoneal surfaces of the intussuscepted part, and it could have been reduced by traction with the greatest ease.

The particulars of a fourth case, which occurred in a young adult man, have been recorded by me in vol. vii, p. 193, of the 'Pathological Society's Transactions.' In this case the patient lived four months from the commencement of his symptoms, and at the post-mortem six inches of the ileum, the entire cæcum, and first part of the ascending colon, were found invaginated within the latter. The coats of the bowel were much thickened, but there were no adhesions, and reduction by traction from within would have been quite practicable.

My experience does not afford a single case at all to be compared with the preceding, in which the patient survived. I treated successfully, by means of injections under chloroform, a case in which I had conjectured that intussusception was present, but the diagnosis did not rest on any certain data, and the stage was a very early one. The patient was a delicate little boy. He had been sick, and had passed slime and blood. I thought that I could feel through the abdominal wall a lump very much like that caused by an intussusception, but there was nothing to be

felt by the rectum. After a free injection under chloroform the bowels acted and the child recovered.

In another case I had a good deal of trouble with a short intussusception about five inches from the anus, which had resulted from the too rapid reduction of an ordinary prolapse of the rectum, seven or eight inches in length. In this instance, after a considerable manipulation, I was successful in effecting a complete reduction.

Thus, it will be seen that at the time the case which is the subject of this paper came under my care my own experience did not supply a single one at all parallel to it in which the patient had been saved; whilst in three all endeavours had resulted in disappointment. In fact, such had been the impression which these cases had made upon my mind, that I had quite determined to resort to operation when next any similar one should present itself.

The case which I have brought before the Society is, so far as I am aware, the first successful one of its kind in English practice. The operation itself, however, is by no means a novelty, and at least three examples of its successful performance are on record.

I may be permitted briefly to refer to the particulars of these.

In one recorded by Velse, and quoted by M. Hévin, in the 'Memoirs of the Royal Academy of Surgery of Paris,' 1784, the patient was a woman aged 50. Intussusception was diagnosed by Nuck, at whose suggestion the operation was performed. The incision was made on the left side of the abdomen, four fingers' breadth from the umbilicus. The intestine was drawn out, and the intussusception was liberated without difficulty, as no adhesions were encountered. The wound was closed and the patient recovered, and lived for twenty years afterwards. In the performance of this operation the intestines were fomented with tepid milk, and the intussuscepted part was well oiled. It is spoken of as having been very easy of performance.

The next case occurred in 1825, and is recorded by Dr. Fuchsius, of Olpe, in 'Hufeland's Journal' (quoted in the

'Edinburgh Medical and Surgical Journal,' July, 1825. Its subject was a man aged 68, who was seen on the sixth day of his illness. In the neighbourhood of the navel, rather on the right side, there was evident hardening and tenderness, which increased and somewhat changed position during attacks of spasm. After five days of further treatment by clysters, &c., the abdomen was opened. An incision was made on the outer edge of the right rectus two inches above the navel. The intussusception was soon found. There were no adhesions, but such difficulty was encountered in effecting reduction that the surgeon decided to open the intestine. This was done by an incision two inches long, admitting of the introduction of the fingers into the intussuscepted part. Reduction was then accomplished, about two feet of bowel being disengaged. The wound in the intestine was stitched up. The patient recovered. The operator recommends, I have no doubt very judiciously, that in future operations the incision should be made in the linea alba, and that, if it be necessary to put stitches in the intestine, they should be cut close off instead of being left with a long end, to come out at the abdominal wound.

A third case of recovery occurred in the practice of an American surgeon, Dr. Wilson, and is recorded in the 'American Journal of the Medical Sciences' for 1836. The patient was a negro aged 20, and the intussusception had lasted seventeen days. There were adhesions, and great difficulty was encountered.

In British practice the operation appears to have been performed only once, and then under very unfavorable circumstances. The patient was an infant only four months old, in whose case Mr. Spencer Wells was consulted, on the fourth day of an intussusception with acute symptoms. The diagnosis was positive, for the involuted portion of intestine could be reached by the finger in the rectum. It was not till the fifth day, when the patient was almost dying, that the parents of the child consented to the operation. The abdomen was opened in the middle line below the umbilicus. The intussuscepted portion was easily found,

but the constriction was so tight that it was not without great difficulty that it was reduced. Its release was at length accomplished, the intestines returned, and the wound closed. The bowel above the constriction being greatly distended with flatus some needle punctures were made for its relief. The child died about five hours after the operation.

As regards other fatal cases after operation, as already implied, I have not been able to find any in English records. Several continental writers refer vaguely to such, and some speak of them as if they had been numerous. I have found a case reported by Carrier, of which the following are the particulars (as given in 'Virchow's Jahresbericht'). The patient was a man aged 23. Pain came on suddenly, and a tumour could be felt in the ileo-cæcal region. On the fifteenth day the abdomen was opened, and an attempt was made to extricate an intussusception which was discovered, but the attempt was unsuccessful. The small intestine higher up was therefore opened. The patient died seven hours afterwards. The post-mortem showed an intussusception of the ileum into the cæcum.

Fatal cases have also been recorded by Max Hertz, Pirogoff, and Gerson. In two of these great difficulties were encountered in freeing the intussuscepted part, and in Pirogoff's case it was found impracticable.

Before attempting further to discuss the propriety or otherwise of this operation I may suitably refer to the symptoms which characterise intussusception, and to some of its natural terminations.

There is a class of cases, and, perhaps, not a very small one, of which the one I have recorded is an example, in which all obscurity as to diagnosis is removed by the discovery of the intussuscepted bowel in the rectum. In all suspected cases this examination should be made. It is quite evident from the descriptions given of the post-mortems in many cases that had the bowel been sought for by the anus it could have been felt. The symptom next in value,

and, indeed, perhaps not second in real importance, is the manipulation of the abdomen and the discovery of the long or oval sausage-like mass which an intussusception constitutes. This is far more easily done than is generally thought possible, especially so with the aid of chloroform. Unless the parietes of the abdomen be fat my impression is that by firm pressure, the patient being under the full influence of an anæsthetic, all doubt as to the existence or non-existence of intussusception, and as to the completeness or incompleteness of its reduction, may usually be removed.

Amongst the other less important symptoms we must mention pain in the abdomen, attacks of spasms, the passage of bloody mucus or of pure blood by stool, the existence, in some cases, of obstruction of the bowels, and in some of almost constant desire to strain at stool. These symptoms will vary much in degree of severity in different cases, and it is of considerable practical importance to remark that the cases may be roughly grouped, much as we do those of hernia, by reference to the tightness of the constriction. We have cases of intussusception accompanied by *strangulation*, and we have others which are *irreducible* only. The former tend rapidly either to the death of the patient, or his relief by gangrene of the constricted part. Their duration is rarely more than a few days. Those, however, in which there is only an irreducible invagination without either stoppage of the contents of the tube or interruption in its blood-supply may run a prolonged course, and they have a greatly diminished chance of spontaneous cure by gangrene. It is in these latter that operative interference is most necessary and has the fairest chance of success. In these the patient may live on for weeks, and the surgeon is permitted a good opportunity both for establishing his diagnosis and proving the inutility of other measures of treatment. The patient's death when it at length arrives is brought about more by exhaustion from long-continued pain than from any inflammatory process. In this class of cases I believe it would seldom be found that the coats of the intestine had become adherent to each other, or that there was any material

difficulty in effecting reduction after opening the abdomen. If the operation were resorted to in cases of *acute strangulation* there would always be the risk that the surgeon might find the parts in a state of gangrene, and might discover that he had interfered only to take away the patient's last chance.

It seems, therefore, of great importance to insist that before attempting the operation the tightness of the strangulation should be estimated.

The diagnosis between mere irreducibility and tight strangulation will usually be easy. In the one there will be severe sickness, constipation, and great general distress tending to collapse, whilst in the other the bowels will continue to act, sickness will be almost wholly absent, and the patient may suffer comparatively little.

I cannot better illustrate this statement than by reminding the Society that in my own case the state of things had existed for a month, and that so slight had been the patient's general symptoms that a surgeon had ordered a cork pad to keep back what he supposed to be an ordinary prolapse.

The same mistake is mentioned as having occurred in several other cases on record.

If in a case of tight strangulation with severe symptoms the patient were seen early and quite before any indications of collapse had appeared, my impression is that opening the abdomen (insufflation, &c., having failed) would be safer than to leave the case to the chance of cure by gangrene, but if the stage were more advanced I think I should prefer to give opium and trust to nature's method.

Before finally deciding as to the need of surgical interference in that class of cases in which, as I have just shown, it is alike most hopeful and most necessary, we must ask what other chances of recovery are before the patient.

Given a case of intussuscepted bowel without sickness and without constipation, therefore, presumably without strangulation, what degree of probability is there that recovery may be obtained either by natural processes or by methods of treatment short of operation? I have just hinted that the

chances of gangrene are not great.[1] The constriction is not tight enough to cause it, and although it must be granted that in a few instances after the bowel has remained for considerable periods in a state of mere incarceration, gangrene does eventually occur, yet it is a rare event; much more commonly the patient sinks from exhaustion. If the chances of recovery by gangrene be but little my impression is that those by spontaneous return of the parts to their natural condition, or their reduction under treatment by insufflation, &c., are much less. At any rate the surgeon will soon know how much he has to hope in either of these directions. I have not found any case recorded in which spontaneous return of a well-recognised intussusception occurred, and those in which art succeeded are comparatively few. It is, of course, the surgeon's duty to give a patient trial to injections, to use fluids and air alternately, and to use them with the patient's body inverted and with the muscles set at complete rest by an anæsthetic, but if he should not succeed quickly by these means it is not likely that he will succeed at all.

[1] Nor must it be forgotten that even when gangrene occurs it does not necessarily lead to recovery. In several cases in the table appended to this paper death followed the expulsion of the detached portion. Dr. Hilton Fagge, in an excellent paper in the 'Guy's Hospital Reports' for 1869, writes as follows upon this point: "Now, as we have already seen in ileo-cæcal intussusception 'expulsion' comparatively seldom occurs, and when it does occur it frequently only postpones the fatal termination instead of entirely preventing it. The patient dies some months afterwards from contraction of the cicatrix, which had formed at the seat of the disease. This appears to me to afford a weighty additional argument in favour of the attempt to explore and pull out an ileo-cæcal intussusception, when the case is directly diagnosed at an early stage, and when inflation has failed to overcome the disease." The precise cause of death suggested by Dr. Fagge is a very probable one, but there are others yet more frequent. A case under the care of M. Fanchon ended fatally three days after the expulsion, there being an abscess at the seat of disease. A case recorded by Dr. Baillie, in which a yard of colon had been passed, resulted in the death of the patient three weeks afterwards. In two other cases death occurred two and four weeks respectively after the sloughing. In another a post-mortem showed a cavity containing fæces, which intervened between the two ends of the bowel; and in another, fever, vomiting, and diarrhœa, preceded death.

Nearly all the recorded instances of success were very recent cases or cases in which the intussusception was small. They serve but little to encourage the surgeon when he encounters such a case as that which I have just recorded. In very few, indeed, was the intussusception long enough to be felt in the rectum, and in scarcely any did success follow after several failures. The opinion of some of our best authorities is so definite on this point that they recommend that all attempts at replacement should be abandoned if they have not succeeded within a short period.

The literature of intussusception is very large, and it is not my intention to trouble the Society with any attempts at its statistical analysis.

My friend and colleague Mr. Waren Tay has, however, kindly collected for me references to a great number of cases, and from these I may be permitted to extract such facts as may seem to bear most definitely on the subject under discussion. We have confined our attention to cases in which the intussusception occurred in the lower bowel.

In the table appended to this report will be found the particulars of numerous cases in which the intussuscepted part presented into the rectum, and either was or might have been felt by the finger. This table must not be considered in any sense exhaustive, but it may yet furnish us with some valuable data.

Of these cases a very few ended in recovery *without gangrene*. In one of these an infant aged eighteen months, in whom an intussusception could be felt by the finger in the rectum, was treated early under the care of Dr. Steele by powerful injections of warm water, and had no return of the symptoms. In a second, a child, under the care of M. Cabaret, had prolapse of bowel from the anus twelve inches in length; whilst at the same time a sound could be passed up for some distance between the rectal mucous membrane and the invaginated parts. Reduction was effected by a gum elastic bougie, which was retained for several hours to prevent relapse. A similar measure was successful in the hands of Dr. Osborne, in a very similar case. It is to be noted that in

all these three cases it would appear to have been the lower part of the colon only which was involved, and it is obvious that in such the chance of success is far greater than when the cæcum or the small intestine comes down.

In the first of the cases of recovery *after gangrene* the patient was a boy aged 6, in whom the early symptoms had been those of strangulation. The bowel appeared at the anus, and about the eighth day a portion, twenty-three inches, came away. In the second case, again, we have symptoms of severe strangulation, and the patient, a girl aged 11, appeared to be at the point of death. As early as the fifth day a portion of colon, cæcum, and mesentery, measuring nearly fourteen inches, was detached.

In the third case the patient was a man aged 40, who voided twenty-eight inches of colon on the fourteenth day.

The fourth case is one of the most interesting on record, from the unusual length of the period before the bowel separated. The specimen was exhibited by Dr. Quain, before the Pathological Society, and the case is recorded in the tenth volume of its 'Transactions.' The patient, a boy aged 5, had suffered for four months from obscure abdominal symptoms, and was finally relieved by the escape of twelve inches of bowel including the cæcum, part of the ileum and part of the colon. He had had irregular constipation and some sickness, but at times his appetite had been voracious. He had never passed blood.

In the fatal cases the influence of early age in accelerating the event seems well marked, a large majority being infants under the age of one year, who died after periods of from one to three days' illness.

It is clear that if, in infants, operative interference is to be of any use it must be resorted to very early. Examination of the cases in which the patient was under two years of age shows that eleven died within two days, five lived as long as the sixth or seventh day, one to the twentieth, and a single very exceptional one survived for a period of nine weeks. This last case is published by Mr. Sidney Jones in the 'Pathological Society's Transactions.' In it the small intes-

tine had travelled through the entire length of the colon, and protruded at the anus until as much as six inches were visible. The child had free action of the bowels, took the breast well, and never vomited. In the first instance, however, severe symptoms of obstruction had been present. Death was finally caused by exhaustion from straining and by the slowly progressing gangrene of the extruded portion. Mr. Jones mentions in his account of the post-mortem a fact of very great importance in reference to the question of operation—that the serous surfaces of the opposed portions of bowel were adherent along their whole extent by firm, fibrous membrane.

In the absence of any data as to the manner in which operations of this kind are borne by very young children we shall probably be right in believing that they are far less hopeful than in those somewhat older. On the other hand it is our duty to remember that the cure by sphacelus, which occurs with tolerable frequency in others, is scarcely ever met with in infants, and that unless rectification is obtained by injections, without much delay, speedy death is almost certain to result.

Very valuable information might be furnished to the surgeon by post-mortem examination as to the feasibility of operative interference in these cases; unfortunately, however, but few of those who have published cases give us specific details on this point. During the last session of the Pathological Society, Dr. Edwards Crisp exhibited a specimen from a child aged eight weeks, with the statement that so tightly was the invaginated part enclosed that it would have been impossible to withdraw it. Mr. Sidney Jones in one case, as just mentioned, found the peritoneal surfaces universally and firmly adherent. In two cases of my own and in one of Mr. Waren Tay's it was found, at the post-mortem, that traction from within the abdomen easily reduced the invagination, and that there was no material damage to the coats of the bowel. In a very considerable number of published cases the details of the post-mortem warrant the belief that an operation would not have been difficult, since no mention

is made either of tightness of constriction, adhesions or gangrene.[1]

One fact disclosed by post-mortem records I may ask especial attention to, and that is the almost uniform absence of peritonitis as a complication. This is specially noted in a great number of cases. In intussusception as in strangulated hernia, and other forms of abdominal obstruction, it may, I think, be taken as an established fact, that unless actual perforation has occurred there will be no peritonitis.

In conclusion, that I may not further weary the Society by the details of isolated facts, I may briefly record my conviction that any one who will carefully examine the evidence for and against will come to the conclusion that operations for the relief of intussusception are not only warrantable, but that in a large number of cases they are urgently demanded.

The cases most hopeful are those in which the symptoms denote incarceration rather than strangulation, and in them the surgeon may take the knife in hand with a good prospect that he will encounter no serious obstacle, and that he will not find either very tight constriction, adhesions, or gangrene. Of the other cases, there are many in which, if the patient be seen early, there is sufficient hope, notwithstanding the severity of the symptoms, to justify the operation, though the surgeon must expect in such to find occasionally that the conditions preclude its completion. Lastly, in a small minority, seen late, or in which the symptoms have from the first been extremely severe, it is probably wisest to

[1] I do not know that we shall gain much by citing the opinions of authors for or against this operation. Amongst many who dissuade us from it are, Dr. Brinton, Mr. Holmes, and Mr. Pollock. On the other side, MM. Rilliet and Barthez, who base their opinion on post-mortem examinations, in which they found reduction very easy, state that "after employing medical treatment during three or four days, and after having made several attempts at inflation, we should not hesitate to perform gastrotomy." Drs. Meigs and Pepper, who quote the above passage, appear to be quite favorably disposed to the operation, and Dr. West's conclusion is to the same effect. Dr. Hilton Fagge, after a careful summing-up of evidence, is a decided advocate of the operation, but suggests it would be well for the surgeon to wait until a case comes before him which is known not to be already of long standing.

decline an operation and to trust to the chance of gangrene.

The following conclusions are appended by way of summary of the facts and statements contained in my paper.

Conclusions.

1. That it is by no means very uncommon for intussusception to begin at the ilio-cæcal valve, and to progress to such a length that the invaginated part is within reach from the anal orifice or even extruded.

2. That it is of great importance in all cases of suspected intussusception to examine carefully by the anus.

3. That in almost all cases of intussusception in children, and probably most in adults, the diagnosis may be made certain by handling the invaginated part through the abdominal wall.

4. That the prognosis of cases of intussusception varies much; first in ratio with the age of the patient; and, secondly, with the tightness of the constriction.

5. That in a large proportion of the cases in which children under one year are the patients, death must be expected within from one to six days from the commencement.

6. That in the fatal cases death is usually caused by shock or by collapse from irritation and not by peritonitis.

7. That in many cases it is easy, by estimating the severity of the symptoms (vomiting, constipation, &c.), to form an opinion as to whether the intestine is strangulated or simply irreducible.

8. That in cases of strangulated intussusception, whilst there is great risk of speedy death, there is also some hope that gangrene may be produced and spontaneous cure result.

9. That in cases in which the intussuscepted part is incarcerated and not strangulated, there is very little hope of the occurrence of gangrene, and it is probable that the patient

will die, after some weeks or months, worn out by irritation and pain.

10. That the chances of successful treatment, whether by the use of bougies or by the injection of air or water, are exceedingly small, excepting in quite recent cases, and that if the surgeon does not succeed by them promptly it is not likely that he will succeed at all.

11. That the cases best suited for operation are those which have persisted for some considerable time, and in which the intestine is only incarcerated, and that these cases are also precisely those least likely to be relieved by any other method.

12. That in the cases just referred to, after failure by injections, bougies, &c., an operation is to be strongly recommended.

13. That the records of post-mortems justify the belief that, in a considerable portion of the cases referred to, the surgeon will encounter no material difficulty in effecting reduction after opening the abdomen.

14. That the circumstances which might cause difficulty are, first, the tightness of the impaction of the parts; secondly, the existence of adhesions; and thirdly, the presence of gangrene.

15. That in selecting cases suitable for operation the surgeon should be guided by the severity of the symptoms, in his estimate of the tightness of the strangulation, and also as to the probability of gangrene having already set in.

16. That in cases in which the patient's symptoms are very severe, or the stage greatly advanced, it may be wiser to decline the operation and trust to the use of opiates.

17. That the operation is best performed by an incision in the median line below the umbilicus.

18. That in cases of intussusception in young infants (under one year of age) the prognosis is very desperate, scarcely any recovering excepting the few in whom injection treatment is immediately successful, whilst a large majority die very quickly.

19. That the fact just referred to may be held to justify,

in the case of young infants, very early resort to the operation.

20. That it is very desirable that all who in the future have the opportunity for post-mortem examination of intussusception cases should give special attention to the question as to whether an operation would have been practicable, and should record their results.

Tabular Statement

Of the Results of different Plans of Treatment, &c., in Cases of Intussusception of or into the Lower Bowel.

The following table has been compiled for me by Mr. Waren Tay, and comprises cases more or less closely similar to the one which is the subject of my paper. We have selected from various sources the recorded examples of intussusception of the bowel, *in which the intestine passed low down into the colon.* We did not wish to include cases in which the small intestine alone was involved, since these, both as regards treatment, symptoms, and probable results, belong to a different category. It was necessary, therefore, to adopt some definite line of limitation, and this we have found in the presence or otherwise of the intussuscepted part in the rectum. It is believed that no cases are included in the following list in which the bowel was not either discovered by the finger or, at any rate, might have been, had an efficient examination been made. It will be seen that this discovery of the bowel by the finger is a symptom of the utmost importance, since it places the diagnosis, both of the nature of the lesion and the part of bowel involved, beyond question.

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
1	Dr. Trevor, Am. Jour. Med. Sci., Jan., 1852, p. 277	12 years, M.	Occasional pain for two or three weeks, then severe pain followed by vomiting; tumour felt in left side of abdomen and on rectal examination; attempts at replacement with stomach tube, etc.	4 days, death	Intussusception of 12 inches of jejunum into the succeeding 12; the mass thickened by inflammation; there was no general peritonitis; the upper end was just below and within the arch and descending colon, and the lower end was firmly impacted in the pelvis; two smaller ones were found of 2 inches and 1 inch in length	Was diagnosed. This was small intestine, yet forced down nearly to anus.
2	Mr. E. Y. Steele, Lancet, 1849, vol. i, p. 680	8 mos., M.	Passage of blood; tumour felt in rectum; slight prolapse; enemata, etc., were without avail	2 or 3 days? death	No post-mortem obtained.	
3	Do.	4 mos., M.	Tumour felt in the rectum; various efforts at reduction made, but without avail	2 days, death	No general peritonitis; the lower end of the ileum, the cæcum, the ascending and greater part of the transverse colon, were invaginated into the sigmoid flexure; the upper two thirds of the innermost portion were of a claret colour, the lower third greenish brown, and in a state of complete sphacelus.	
4	Do., Lancet, 1859, March 19, p. 287]	18 mos., F.	Tumour felt in the rectum; "a considerable length of inverted gut was occupying the rectum;" there was a watery discharge tinged with blood; the case was treated early by powerful injection of warm water and subsequent cautious narcotism	6 to 12 hours, recovery	There was no feeling perceptible as if the intussusception had suddenly given way, but the gut did not again bulge down into the rectum	An instance of success by enemata.

5	Cabaret, Rev. de Ther. Med. Chir., 13, 1858, Schmidt's Jahrbüch., 101, 1859, p. 322	2 years, M.	Constipation after diarrhœa, then prolapsus which could be replaced; on seventh day a sudden prolapse, which could not be reduced; on the eighth day twelve inches of colon were prolapsed, while a sound could be passed a long way upwards by the side of it	7 days, recovery	Attempts to reduce with the fingers only were without avail; Le Pelletier's method was successful; pressure with a gum-elastic sound, which was not removed finally till the third day, as the prolapse recurred a few minutes after the pressure was withdrawn	Probably the prolapse was of rectum chiefly. Compare with the first mentioned, on page 39.
6	Dr. Worthington, Am. Journ. Med. Sciences, Jan., 1849, p. 97	3½ years	For two years had had various intestinal symptoms; for six weeks had diarrhœa and protrusion of bowel at the anus	6 weeks? death	The cæcum was invaginated along the whole length of the colon and rectum, carrying with it the lower portion of the ileum and the first part of the colon; more than 2 feet of bowel had been inverted; the cæcum must have passed through the sphincter in the child's efforts to evacuate the bowels.	
7	Mr. T. Blizard, Med. Chir. Trans., vol. i, p. 169, 1812	5 mos., M.	A tumour about the size of an egg on the left side of the abdomen	4 days, death	The lower end of the ileum, the cæcum and its appendix, the ascending colon, &c., were invaginated into the sigmoid flexure and rectum to within 1½ inch of the anus; there was no peritonitis; the invaginated parts were gangrenous; "they might have separated had the child's constitution not given way."	
8	Mr. Langstaff, Edin. Med. Journ., July, 1807, p. 263	3 mos., ?	Vomiting; passage of blood and mucus; hard tumour felt on the left side of the abdomen; prolapsus	5 days, death	The end of ileum, the cæcum, and colon invaginated into rectum; there was also a smaller invagination in the opposite direction.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
9	Penquier, L'Union Médicale, Aug. 22, 1861; Dr. Smith, Amer. Journ. Med. Sci., Jan., 1862	4 mos., M.	Passage of blood; tumour felt five or six inches from the anus, and a hard elongated tumour in the left iliac fossa	2 days, death	Cæcum and its appendix, ascending and transverse colon into descending colon, close to rectum. It is stated that invagination of the large intestine is common among children in Brittany, where the practice prevails of bandaging them tightly in linen and leaving them alone for some hours.	
10	Dr. Carter, Lancet, June 9, 1849, p. 607	4 mos., F.	A large sausage-like body felt in left iliac region; enemata only reached a certain point	6 days, death	Invagination found in left iliac region, but not described	Probably could have been felt per anum.
11	Mr. Nind, Lancet, 1849, vol. i, p. 681	4 mos., M.	One sanguineous stool	4 days, death	Cæcum and ascending colon in sigmoid flexure. "The invagination was so complete that from the congestion, &c., which had occurred I could not reduce it till the enclosing portion of the gut was divided nearly in its whole length;" it "was of a deep purplish colour, with small ash-coloured patches of gangrene."	Ditto.
12	Dr. Edwards, Med. Times & Gaz., 1861, vol. ii, p. 531	3½ years, M.	Pain and swelling of right side of abdomen, which passed away eight months previously; prolapse, which could be returned four months previously; for two days the prolapse became permanent	4 days? death	*Post-mortem.*—A portion of bowel 2½ inches in length protruded from the anus; a large mass could be felt in the left side of the abdomen; the cæcum and colon invaginated into descending colon, sigmoid flexure and rectum; the part protruding	

					from the anus consisted of the inverted cæcum and its appendix, and the opening of the ileo-cæcal valve was visible on the prolapsed part.	
13	Mr. Ash, Med. Times & Gaz., 1867, vol. ii, p. 505; Brit. Med. Journ., 1868, vol. i, p. 117	6 years, F.	A tumour could be felt within the anus, and finally a prolapse occurred; attempts to push the tumour back unsuccessful	10 days, death	A post-mortem was obtained, but no description of the state of parts is given; it is called an intussusception of the rectum.	
14	Mr. Young, Brit. Med. Journ., vol. ii, p. 779, 1859	9 mos., M.	A tumour could be seen and felt less than an inch from the anus	28 hours, death	*Post-mortem.*—Ileum, cæcum, and colon invaginated into colon and rectum.	
15	Dr. Philipson, Brit. Med. Journ., Sept. 24, 1864	10 mos., M.	Tumour felt about four inches from the anus; repeated attempts at reduction (digital, enemata, and insufflation) unsuccessful	28 hours, death	Ileum, cæcum, and colon, invaginated into colon and rectum, the lowest portion of the tumour being the part near the ileo-cæcal valve. "An attempt was made to reduce the tumour in the same manner as during life, but was quite unsuccessful. Traction was then made on the small intestine, and it was only dislodged after a considerable amount of force." "The cæcum and appendix were then drawn out, but still more force was required; the adhesions were very firm."	
16	Dr. Merriman, Lancet, 1844, vol. ii, p. 298	Child	Not given	4 days, death	Cæcum, appendix, and ileo-cæcal valve invaginated into colon	Probably might have been felt if examination per anum had been made during a straining fit.

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
17	Mr. Snow, ibid.	Child	Not given	Death	Ileum into sigmoid flexure	Same as No. 16.
18	Markwick, Lancet, 1846, vol. ii	4 mos.	Blood passed; the symptoms began soon after birth	4 mos.? death	The colon was intussuscepted into the sigmoid flexure; it was impossible to withdraw it	Ditto.
19	Clark, Lancet, 1849, vol. ii, p. 206	6 mos.	Left side of abdomen hard and prominent	2 days, death	Ileum, cæcum, and colon intussuscepted into descending colon	Ditto.
20	Burford, Lancet, Oct. 31, 1840	6 mos.	Passage of blood	3 days, death	*Post-mortem.* — "Was surprised to find the rectum fall." Ileum, cæcum, and colon invaginated into sigmoid flexure and rectum.	
21	M. Robin. M. Hévin's Memoir in Mém. de l'Acad. Roy. de Chir., t. xi, p. 324	3½ years	Prolapsus after suffering more or less for three months	6 days, death	Cæcum and greater part of colon in the rectum. "It was found impossible to withdraw the invaginated intestine, it had contracted strong adhesions."	
22	M. le Blanc. M. Sabatier's Mémoire (Mém. de l'Acad. Roy. de Chir., t. xv, p. 35)	A child	Prolapse six or seven inches in length	15 days, death	Ileum, cæcum, and colon, were invaginated into the rectum; it was impossible to reduce this.	
23	M. Puy, quoted by M. Sabatier (l. c.)	40 years, M.	Prolapse to the extent of about six inches; the first attack occurred about two months before death, but he apparently recovered from this	16 hours, death	Ileum and colon invaginated into the rectum.	

24	MM. Roux et Lavernet, quoted Dict. des Sciences Med., vol. xxiii, p. 560	—	—	Death	The sigmoid flexure of the colon was invaginated into the rectum to the extent of 13 inches.	
25	Dr. Ash, quoted by Hunter, Trans. Soc. for Imp. Med. Chir. Knowledge, vol. i, p. 108	9 mos., M.	Discharge of blood; tumour felt in the left iliac fossa	60 hours, death	Ileum, cæcum, and colon invaginated into colon and rectum.	
26	Monro, sen., Morbid Anat. Alimentary Canal; Dr. Smith, Am. Journ. Med. Sci., Jan., 1862	4 mos., M.	Slime and blood discharged	68 hours, death	Ileum and colon into colon and rectum.	
27	Mr. Clarke, Lancet, Feb. 10, 1838 (Gorham, Guy's Hosp. Rep., Oct., 1838, p. 331)	11 mos.	Hæmorrhage	62 hours, death	Ileum, cæcum, and colon into colon, close to sigmoid flexure	? Could have been felt.
28	Dr. Baer, Am. Jour. Med. Sci. (Gorham, l. c.)	16 mos.	Hæmorrhage, and large tumour felt in left iliac region	48 hours, death	Ileum, cæcum, and colon into sigmoid flexure.	
29	Mr. Cunningham, Med. Gaz. Sept. 15, 1838 (Gorham, l. c.)	9 mos.	Hæmorrhage, and tumour felt in left iliac region	40 hours, death	Ileum, cæcum, and colon in sigmoid flexure and rectum.	
30	Mr. Whately, Phil. Trans., vol. lxxvi, p. 305	M.	"The valve of the colon at last got as low as the anus, and when he went to stool he only emptied the ileum"	Death	Ileum, cæcum, and colon in sigmoid flexure and rectum.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
31	Dr. O. Ferral, Dub. Path. Soc., Lond. Med. Times, Jan. 16, 1847 (Dr. Smith, l. c.)	12 mos., M.	Intense pain, passage of mucus and blood	6 days, death	Ileum and cæcum into colon; there were two orifices, one leading into the ileo-cæcal valve, the other into the appendix	Probably could have been felt.
32	W. S. Partridge, Prov. Med. and Surg. Journ., May 3, 1848 (Dr. Smith, l.c.)	4 years	Passage of blood and mucus	3 days, death	Ileum, cæcum, and colon invaginated into rectum	Ditto.
33	Dr. Harland, Med. and Phys. Res., Philad., p. 565 (Dr. Smith, l. c.)	5 mos., F.	Prolapse	Not stated, death	Ileum, cæcum, and colon into colon and rectum; the cæcum protruded from the anus.	
34	Mr. Davies, Med. Repos., Dec., 1824 (Dr. Smith, l. c.)	6 years, F.	Diarrhœa, passage of mucus and blood	8 months, death	Ileum, cæcum, and colon into colon and rectum	Probably could have been felt.
35	Dr. Kennedy, Dub. Journ. Med. Science, March 4, 1844 (Dr. Smith, l. c.)	4 mos.	Passage of blood	1 day, death	Ileum, cæcum, and colon were invaginated	Ditto.
36	Mr. Perrin, Lancet, March 26, 1853 (Dr. Smith, l. c.)	3 mos.	Bloody stools	2 days, death	Ileum, cæcum, and colon, in descending colon and sigmoid flexure	Ditto.
37	Dr. Smith (l. c., Case 34)	3 mos., M.	Passage of mucus and blood; prolapse twelve hours before death; distension in right iliac region	7 days, death	Ileum, cæcum, and colon into colon, &c.	

38	Levi Gaylord, Am. Journ. Med. Sci., October, 1827 (Dr. Smith, l. c.)	6 years, M.	Vomiting, constipation, then an evacuation; prolapse after seven or eight days, and the next day 23 inches separated	9 days, recovery	Portion of intestine not stated	Recovery by sloughing.
39	Jacobi (Dr. Smith, l. c.)	—	Tumour felt two or three inches from anus	Death	An invagination was found in the rectum.	
40	Monro, Edin. Phys. and Lit. Essays, vol. ii, p. 386	18 mos., M.	Prolapse; after reduction a tumour could be detected with an opening at the lower part like an os tincæ; large enemata were given, and a long probe of whalebone armed with sponge was used, but without success	A few days, death	The invagination began just below the upper part of the sigmoid flexure.	
41	Mr. Stanley, Lancet, March 11, 1826, p. 813	Middle age, F.	Prolapsus	Death	A surgeon gently pulled at the intestine, and a yard and three inches came away; it proved to be a portion of ileum.	
42	Mr. Howship, Ed. Med. & Surg. Journ., April, 1812	4 mos., F.	Passage of blood; injections failed	6 days, death	The lower part of the colon and the upper part of the rectum were invaginated into the rectum. He regrets "that this was not felt during life."	
43	Mr. J. W. Bowman, ib., Oct. 1813	11 years, F.	Obstruction of the bowels and every symptom of approaching dissolution	5 days, recovery	Portion of colon, cæcum, and mesentery, measuring 19¾ inches, passed by stool	? Could probably have been felt. Recovery by gangrene.
44	Mr. Valentine, ib., April, 1826	40 years, M.	Passed 28 inches of colon	14 days, recovery	—	Ditto, ditto.
45	Mr. Sydney Jones, Path. Tr., vol. viii, p. 179	4 mos., M.	Passage of mucus and blood; improvement for three weeks; at the end of forty-six days, prolapse; this increased till as much as 6 inches protruded	9 weeks, death	The lower part of the ileum had passed along the whole length of the colon and out at the anus; it was still pervious; there were evidences of progressive sloughing.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
46	Mr. Holmes, Path. Trans., vol. viii, p. 177	40 years, M.	Tumour felt at a distance of about half an inch from the anus	10 days ? death	The sigmoid flexure of the colon and the upper part of the rectum were invaginated. In the middle of the ascending colon was a ragged opening, from which fæces had escaped.	
47	Mr. Ballard, ib., p. 185	6 mos., F.	Blood-stained fluid passed per anum	4 days, death	Ileum, cæcum, and colon invaginated into rectum within two inches of the anus.	
48	Dr. Quain, ib., vol. x, p. 160	5 years, M.	Pain in region of bladder; no passage of blood; sickness and constipation	4 mos., recovery	8 inches of the ileum, the cæcum, and 4 inches of the colon were passed by the anus	? Could probably have been felt. Recovery by sloughing.
49	Dr. Buchanan, ib., p. 171	7 mos., F.	Passage of blood; physical examination of the abdomen negative	53 hours, death	Ileum, cæcum, and colon invaginated into sigmoid flexure and rectum to within half an inch of the anus. The intestine could be withdrawn without special difficulty	Stress is laid on the importance of an anal examination.
50	Mr. Nunneley, ib., vol. xi, p. 109	3 years, M.	Passage of a little bloody mucus	11 days, death	A foot of ileum had passed through the ileo-cæcal valve into the large intestine; there was no appearance of inflammation whatever, neither lymph nor blood was effused	? Could have been felt.

51	Dr. Lettsom, Phil. Trans., vol. lxxvi, p. 305	4 years, F.	No special symptoms noted; child not seen for three weeks	3 mos., death	*Post-mortem.*—A finger introduced into the anus detected a round substance in the rectum, with an opening in the middle, not unlike an os-tincæ; the finger passed completely round this between it and the wall of the rectum; the enclosed intestine was in a state of commencing gangrene, but could be easily withdrawn; a portion of ileum contained was uninverted, so was the appendix cæci.	
52	Dr. Osborne, Aitken's Medicine, vol. ii, p. 814	A child	Tumour felt in the rectum; at the end of thirty-four hours it almost presented at the anus; an elastic bougie was passed into the orifice and pushed up; it carried the intestine with it, but "more owing to straightening of the canal than any force used"	2 days ? recovery	—	Recovery after reduction by a bougie.
53	Dr. Thomson, Edin. Med. Journ., pp. 300 and 316. From an Italian source	40 years, F.	After seven or eight days colic symptoms; passage of part of colon, the cæcum and its appendix; a month later there were still colicky symptoms, and a hard, circumscribed tumour could be felt in the left iliac region	50 days, death	When the omentum was raised, two openings were found in the colon, one of which received the ileum and its mesentery; the omentum performed the part of an outer coat, so that no fæces escaped from the intestine. The rectum "appeared full of fæces, but on being cut up the ileum and mesentery, for a Parisian foot, were found pushed into the colon as far as the rectum."	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
54	Dr. Greig, Edin. Med. Journ., Oct., 1862, p. 312	4 mos., M.	Passage of blood; tumour in left side of abdomen; an enema could not be thrown up	42 hours, death	Ileum, cæcum, and colon invaginated into rectum to within an inch of the anus. "After removal of the tumour the cæcum was easily drawn out of the colon, and restored to its natural position, but the greatest difficulty was experienced in getting the swollen, small intestine reduced through the ileo-cæcal valve, which seemed even then to be in a spasmodic condition"	Dr. Greig narrates four other cases which recovered under the use of inflation, but in these the tumour was on the right side, probably short intussusceptions. In one of them he had to inflate several times.
55	Dr. Greene, Brit. Med. Journ., March 18, 1871, p. 278	4½ mos.	A tumour, much resembling a small sausage in shape and density, was felt on left side of abdomen; passage of slime	Several days, death	Ileum, cæcum, and colon into descending colon and sigmoid flexure.	
56	Dr. J. St. C. Gray	5 years, M.	Tenesmus, passage of blood at first, then of blood-stained mucus. "There was considerable tympanites, but nothing was ascertained tending to throw light on the case, either by percussion or by examination of the rectum by the finger." Turpentine enemata, then various remedies	8 days, death	There was no trace of peritonitis; colon was invaginated into colon and upper part of rectum; the cæcum was not involved	The distance from the anus is not stated, but possibly by pressing on the abdomen, and examining during straining, the finger might have reached the "upper part of the rectum."
57	Mr. King, Lancet, June 17, 1854	6 years, M.	Suddenly seized with symptoms of ileus; in four days convulsions and insensibility	11 days, recovery	The cæcum and appendix, with part of the ascending colon, passed per anum; afterwards the right lower extremity became swollen and gangrenous, the leg separating at the knee-joint	Recovery after gangrene.

58	Hennet, Pr. Ver. Ztg. n. F., 1, 31, 1858. Schmidt's Jahrb., 101, 1859, p. 321	Adult, M.	Sudden passage of blood in vomit and by anus, constipation, and pain; clysters of no avail; petechiæ on various parts of the body; great prostration. On the 9th day some improvement. On the 12th day fæces passed. 13th day the man drew attention to a mass protruding from his anus	12 days, recovery	The cæcum and the vermiform appendix were passed per anum; the gut was in sufficiently good condition for the longitudinal bands, &c., to be made out; the man was a mere skeleton at the time the sphacelation occurred, and though he immediately began to improve, it was six months before he resumed his duties as a soldier	Recovery by gangrene.
59	M. Sobaux. (M. Hévin, Mém. de l' Acad. Roy. de Chir., t. xi, 1784)	Adult, M.	A portion of colon, 23 inches in length, passed per anum after about three weeks' illness.	3 weeks, recovery	—	Ditto.
60	M. Fauchon. (M. Hévin, l. c.)	48 years, M.	The whole of the cæcum, with 6 inches of the colon, and the same length of ileum, were passed after twenty-five days' illness; the patient seemed well afterwards, but died three days later	28 days, death	An abscess communicating with the gut was found	Death after gangrene and separation of gut.
61	Dr. Thomson, Edin. Med. Journ., 1835, p. 301	40 years, M.	The man was thrown down and trampled on; after some weeks he suddenly felt something in the rectum; at last protrusion occurred, and when he laid hold of the mass extruded he pulled away intestine	Some weeks, recovery	The portion of intestine was a span in length, and at one end was found the ileo-cæcal valve	Recovery by gangrene.
62	Dr. Thomson, l. c., p. 304	11 years, F.	Very severe abdominal symptoms; threatened dissolution	5 days, recovery	A portion of the colon, the cæcum and mæso-colon measuring 13¾ inches, was passed by the anus	Ditto.

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
63	Dr. Thomson, l. c., p. 305	24 years, M.	Around the umbilicus was an oval swelling larger and longer than a turkey's egg	40 days, recovery	The whole of the cæcum with its appendix was discharged per anum	Recovery by gangrene.
64	Dr. Thomson, l. c., p. 308; also 1836, p. 374	4½ years, M.	Colicky pains in the belly, with passage of blood; protrusion of intestine from anus five days before its separation	Some days, recovery	The boy, while suffering from smallpox, passed the whole of the cæcum with its appendix, and part of the ileum	Ditto.
65	Dr. Baillie, Trans. Soc. Improvement of Med. Knowledge, vol. ii, p. 144. (Dr. Thomson, l. c., p. 312)	50 years, F.	Frequent passage of blood and vomiting; diarrhœa came on and lasted for many days	"Many days," death	No autopsy. A yard of colon was passed three weeks before death	Death, although the gangrenous bowel had passed.
66	Dr. Thomson, l. c., p. 313	30 years, F.	Colic, fever, stercoraceous vomiting. On the 8th day the cæcum and a part of the colon were passed	Death	She died a fortnight after the separation of the bowel in connection with a confinement of a stillborn child; the whole abdomen was filled with purulent serum	Ditto.
67	Dr. Thomson, l. c., 1836, p. 378	40 years, F.	After various abdominal symptoms, passage of a membranous substance	Recovery	It was considered to be the cæcum which was passed	Recovery by gangrene.
68	Dr. Thomson, l, c., p. 378	35 years, M.	Dysenteric symptoms; about 18 inches of colon passed	15 days, recovery	—	Ditto.
69	Dr. Thomson, l. c., p. 380. From Meckel	17 years, F.	"A fever, attended at first with constipation, and then with diarrhœa"	4 weeks, death	Separation of cæcum and appendix, and later, of whole of transverse and ascending colon with portion of ileum, thirteen inches in length, "so far intus-	Death long after gangrenous detachment of the bowel.

					suscepted as to protrude from the anus." The patient did not die till eight weeks after the beginning of the illness, and four weeks after the separation of the intestine.	
70	Dr. Thomson, l. c. p. 380	7 years, F.	Fever and pains in the abdomen; in the region of the loins was felt a pretty hard swelling of the size of a goose's egg; at length the whole cæcum with its vermiform appendix was discharged per anum	Death	At first the patient improved after passage of the intestine, but was at last carried off by fever, colic, vomiting, and diarrhœa. The cæcum and part of the ileum had evidently been invaginated into the colon, and had sloughed off	The cause of death is not stated. Death occurred after gangrenous separation.
71	Hill, Month. Jour. Med. Sci., vol. v, 1845, p. 572. (Dr. Peacock, Path. Tr., xv, p. 122)	65 years, F.	Constipation, vomiting, diarrhœa; constipation for a week; then in five days passage of *forty-four* inches of intestine; forty days later she sank, exhausted	53 days, death	The portion passed proved to have been the sigmoid flexure; only fourteen inches of colon remained and terminated in a cavity containing fæces from which the rectum arose.	
72	Dr. Peacock, l. c. p. 122	67 years, F.	Pain followed by diarrhœa; a large portion of the rectum and colon was passed on the fifteenth day	15 days, recovery	She ultimately recovered, but suffered from pain in the abdomen.	
73	Dr. Hunter (Jedburgh), Lancet, Mar. 9, 1872	9 mos.	Was not very fretful nor peevish; took the breast well till near the end; was very thirsty, and drank eagerly of water; vomiting was almost constant, though it was not fæcal at any time. For the last fortnight the ileo-cæcal valve protruded at the anus often to more than an inch beyond, and at other times lay up in the rectum when put up	20 days, death	The invagination had begun at the ileo-cæcal valve.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
74	Wilson, Clinical Record, Feb., 1870. (Virchow's Jahresb., 1870, Bd. ii, Abth. 3)	4 mos.	—	7 days, death	Ileum, cæcum, and colon invaginated into the colon and upper part of the rectum.	
75	Wagner, Jahrb. f. Kinderhlk., n. f. iii, 343	2 years, M.	The tumour was felt on rectal examination; after inflation the tumour at once disappeared	Recovery	The child remained well at the end of eighteen months	Recovery after inflation.
76	Ditto	4 years, M.	Tumour on the left side of the abdomen; none felt on examination per anum	Recovery	Clysters had no effect. After eight pumpings with bellows a loud report was heard; the next day there were signs of the tumour returning, but the whole disappeared after inflation. The child remained well	Ditto.
77	Dr. S. Wilks, Lancet, May 21, 1870	6 mos., M.	Sickness; blood by rectum; lump felt to left, above umbilicus, which hardened on pressure. On passing the finger into the rectum a round projection could be felt four inches up with a circular orifice in the centre; inflation was used and the tumour disappeared	24 hours, recovery	"Ileum into cæcum probably." The child remained well for about a fortnight, was then brought with a lump again to be felt and had passed blood. The mother declined further treatment, and the case was lost sight of	The result remained doubtful.
78	Dr. Hilton Fagge, Guy's Hosp. Rep., 1869, p. 289	5 years, M.	Pain and abdominal tumour the only symptoms for two (? four) months; symptoms of strangulation with hæmorrhage four days before death. Peculiar feeling of hardness whenever tumour was grasped; only half	4 months, death	The ileum, cæcum, and colon into descending and sigmoid colon. Shreds of lymph (adhesions) of no very recent formation united the parts together; no ulceration nor gangrene; the finger could easily be passed along the entering	

				a pint of gruel could be thrown up		bowel which was not strangulated. "The inner layers of the bowel were but little inflamed, and were far from having commenced to slough off."	
	79	Ditto, p. 302	1 year	Had passed blood; twelve inches of intestine (ileum, cæcum, and colon) sloughed	Recovery	—	Recovery by sloughing.
	80	K. v. Mosengeil, Arch. f. Klin. Chir., xii, p. 75	7 mos., M.	Doubtful symptoms for fourteen days; then for four days constipation, straining, and propulsion of a tumour nearly to the anus; next day prolapsus; this increased on following day, and the whole was replaced by a catheter. The child appeared well. In a few hours the protrusion returned and no efforts were successful in reducing it. Three days later some mucous membrane sloughed; the next day an artificial anus was formed in the left groin, and an intussuscepted portion of gut found in the portion opened. A catheter could be passed a long way up by the side of the invaginated portion. On the third day afterwards the child died	27 days, death	At first, injections of water and the use of a sound did not succeed, then the latter replaced the intussusception; but later in the same day nothing was of any avail. No post-mortem. The operation performed was of no avail. The operator recommends that in future the incision should be made on the right side. He would have done so here afterwards, but the child was too exhausted	The question of performining abdominal section and replacing the intestine does not seem to have been entertained. It would appear to have been a favorable case. The case is exceptional in respect to the long survival of a very young child.
	81	Dr. Steffan. (Dr. Pilz, Jahrb. f. Kinderheilk., n. f., iii, p. 6, 1870)	3 mos., M.	Well-marked symptoms	4 days, death	*Post-mortem.*—Ileum, cæcum, and colon into descending colon; the folds were covered with blood-stained mucus and what seemed to be layers of fibrine	Cited on account of condition at post-mortem.

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
82	Dr. Ecker (ibid.)	6 years, M.	Well-marked symptoms; passage of portion of small intestine; yet, on the fourteenth day, sudden symptoms of peritonitis	Death	No post-mortem	Cited as fatal result after sphacelus.
83	Dr. O. Groos. Berlin. Klin. Woch., 1870, p. 395. (Œsterr. Jahrb. f. Päd., 1871, Bd. ii, p. 58.)	6 mos., M.	After twenty-four hours tumour in left abdomen and in rectum; third day prolapsus; attempts with finger and with clysters without success; repeated second day	5 days, death	*Post-mortem.*—Peritonitis. Old adhesions of parts of ileo-colic intussusception to each other. The author remarks that the intussusception must have been of old standing, and that the symptoms came on when the passage previously existing was blocked by the subsequent invagination of small intestine into it.	
84	Dr. Max. Herz (l. c. 1872, Bd. i, p. 5)	7 mos., F.	Sickness and constipation; clysters thrown up on third and fourth days; on fifth day blood and slime passed; no tumour; injection of air; sixth day tumour left side of abdomen and in rectum; on the seventh day convulsions	6 days, death	No post-mortem. See also case of abdominal section.	
85	Dr. Faber, Wurtemb. Corresp. Blatt., No. 25 (Virchow's Jahresb., 1870, Bd. ii, p. 160)	11 years, M.	Tumour in abdomen just under navel; passage of blood. The tumour was long and tolerably hard	Recovery	After four injections of cold water at different times the tumour disappeared	? Examined by rectum.

86	Van Nes, Schmidt's Jahrb., Bd. lvii, 1848, p. 59 (No. 42, Pilz*) (Jahrb. f. Kindhlk., n. f., Bd. iii, 1870)	5 mos., M.	Blood-stained mucus	7 days, recovery	[* This with the others so marked are given by Pilz as cases of intussusception into or beyond the rectum, in which recovery resulted, and for that reason we quote them. He gives no details. We have obtained particulars of the others; they amount to eight recoveries out of forty of such cases. All are given here.]	The account in Schmidt's Jahrb. seems doubtful.
87	Gelmo, Jahrb. f. Kinderhlk., Bd. v, p. 175	8 mos., M.	Seen on fourth day; injections of water of no use; fifth day, air no use; intussusception descending; sixth day, tumour felt in the rectum; after seven injections of water the tumours disappeared	5 days, recovery	On the fifth day calomel was given in two-grain doses every half hour, *i. e.* ten grains altogether. The author remarks that his case was interesting, because "in spite of the five days' duration, no adhesion of the surfaces had occurred"	In this case perseverance with water injections succeeded after insufflation had failed.
88	Nissen, Ficke und Opp. Zeitschr., Bd. xix, p. 162 (78 Pilz) *	9 mos., F.	Tumour on left side of abdomen; prolapsus; reposition with a sound and injections of water	9 days, recovery		
89	Neumann, Inaug. Diss. Halte, 1842 (79 Pilz)*	9 mos., M.	Colon into descending colon; tumour felt by the rectum; sound used	3 days, recovery		
90	Legoupil, Gerson Magazin. (120 Pilz) *	4½ years, M.	Ileo-colic; prolapse; blood passed	30 days, recovery		
91	Prestat, Journ. f. Kinderkrank., 1863, Bd. xli, p. 310	9 years, F.	Symptoms of intussusception, then prolapse, which was replaced, but the child soon afterwards passed a mass of intestine	7 days, recovery	Sphacelus on eighth day; the lower end of the small intestine with a fold of mesentery. The child was under observation for a year afterwards.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
92	Gieffers, Caspar's Woch., 1815 (156 Pilz)*	13 years	Prolapsus	Recovery	Sphacelus of twelve inches and a half of large intestine	Recovery by gangrene.
93	Van Nes, Schmidt's Jahrb., 1848, Bd. lvii, p. 59	9 mos., M.	Tumour felt in rectum and in abdomen. "After three days symptoms of peritonitis"	3 to 4 days, death	Transverse colon into descending colon and rectum. After death he opened the abdomen on the left side, in front, and then the intestine. He found a long invagination and could not reduce it; he also found evidences of peritonitis.	
94	Dr. H. A. Beach, Boston Med. and Surg. Journ., Nov. 5, 1868	24 years, M.	Tumour felt in rectum after injections had been given and the lower part of the rectum cleaned out	85 hours, recovery	He placed the man "on his shoulders and knees" and gave injections. The intussusception slipped up, but returned when the man moved about four hours afterwards. It was again driven up	Recovery by injective treatment.
95	W. Pepper, Phil. Med. Times, Sept. 1, 1871 (Virchow's Jahresb., 1871, Bd. ii, p. 152)	6 mos.	—	4 days, death	Invagination of ileum, cæcum, and colon into colon.	
96	Cooke, New York Med. Record, May 1, 1871	An adult ?	Constipation; passage of slime for three or four days, then an intussusception detected in the rectum	5 days, recovery	The tumour was pushed up as far as possible, then driven still farther by injections while the patient was on knees and elbows. The next day the injection was repeated and an evacuation followed	Recovery by injections.

97	Kjelberg and C. Blix (Virchow's Jahresb., 1871, Bd. ii, p. 606)	11 mos., F.	Tumour felt in abdomen and by rectum; attempts at reduction fruitless	Death	Invagination of ileum and colon into descending and sigmoid colon.	
98	Gaetano Moretti, Annali Univ. di Med. Giugno (Virchow's Jahresb., 1871, Bd. ii, p. 153)	40 years, M.	Symptoms of invagination; prolapse of sixteen inches of intestine; it could easily be pushed back a certain distance, but no farther; finally, it came away altogether	Recovery	The portion of intestine which came away was sixteen inches long, and was believed to belong to the sigmoid flexure of the colon. The patient remained under observation for two months, and was then quite well	Recovery by gangrene.
99	Dr. Hodges, Boston Med. and Surg. Journ., Aug. 6, 1868, p. 5	3 years	After various symptoms of colic, vomiting came on, and tumour felt in right iliac fossa; none in rectum. Two days later blood passed, and the next day a tumour was found in the rectum. On the following day the child was sent to hospital. Tumour felt in rectum, and made visible by the use of a speculum. Child in a moribund condition. A steel sound was used, and the tumour pushed out of sight, but the child died eight hours later	4 days? death	*Post-mortem.*—The cæcum was found to present. No statement as to peritonitis	Tumour made visible by use of a speculum.
100	Hachmann, Zeit. f. gesamt. Med. v. Fricke u. Oppen., Bd. xiv, p. 289 (No. 1 Pilz)	11 weeks, M.	Passage of blood; tumour in abdomen and in rectum	5 days, death	Ileum, cæcum, and colon into colon as far as the rectum.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
101	L. Smith, N. York Path. Soc., 1861 (No. 8, Pilz)	3 mos., M.	Passage of blood; tumour in abdomen and in rectum	8 days, death	Ileum through the colon.	
102	Plath, Caspar's Woch., 1839, p. 432 (No. 10, Pilz)	16 weeks, M.	Passage of blood; tumour in abdomen and in rectum	2 days, death	Cæcum and colon into descending colon and rectum; there was no gangrene. The intussusception, it is said, could not be drawn out, owing to constriction at its commencement.	See No. 121.
103	Basedon, Siebold Journ. f. Geburtsk., Bd. vii, p. 512 (No. 26, Pilz)	4 mos.	Passage of blood; tumour in rectum	2 days, death	Colon ascendens in colon descendens.	
104	Dr. Staug, Journ. fur Kinderkrank., 1863, Bd. ii, p. 130	5½ mos., M.	Passage of blood-stained mucus; tumour could be felt in the abdomen, and could be seen through the distended anus; a sound was used and clysters were thrown up without effect	4 days, death	Cæcum and colon into colon (transverse). There was a plastic exudation on the layers of intestine in the intussusception, but it did not amount to much; the layers were cut open.	
105	Schwarzwelder, Gaz. Hebdom., 1857, p. 583 (No. 73, Pilz)	7 mos., F.	Blood-stained mucus passed; prolapse; tumour in abdomen noticed	6 days, death	Ileum, cæcum, and colon into colon and rectum; no peritonitis. (Quoted in Gaz. Hebdom. from the Cincinnati Med. Observer, July, 1857, p. 295; case under the care of Mr. Wilson.)	
106	Husch, Caspar's Woch., 1838, p. 647 (No. 81, Pilz)	9 mos., F.	Blood passed; prolapse; the cæcum was outside the anus	30 hours, ?	Ileum, cæcum, and colon into rectum. There were no adhesions nor any exudation.	

107	Dr. Thorowgood, Med. Times and Gaz., 1861, vol. ii, p. 160	9 mos., F.	Blood-stained mucus passed; a large, elongated tumour felt on the left side of the abdomen	4 days, death	Ileum, cæcum, and colon into descending colon; no adhesions, no softening, nor gangrenous appearance of any kind.
108	Mr. Matthias Rowe, Lond. Med. Gaz., vol. xv, October 25, 1834	10 mos.	Passage of blood-stained mucus; tumour felt in rectum	30 hours, death	The ileum, cæcum, and colon in the rectum. A drawing of the parts is given. "It was with difficulty that the colon could be drawn out of the rectum."
109	Augustin, Diss. Inaug. Halle, 1836 (No. 103, Pilz)	2 years, M.	Blood-stained mucus; tumour in abdomen and in rectum	11 days, death	Colon transverse, and descending in the rectum.
110	Abercrombie, Diseases of the Stomach, p. 123	2 years and 5 mos., M.	Vomiting, pain, passage of bloody mucus and blood; prolapse on second day; the caput coli protruded	2 days, death	Ileum, cæcum, and colon into colon. There was congestion, no adhesions; the ileum was tolerably healthy.
111	Neumann, Diss. Inaug. Halle, 1842 (No. 109, Pilz)	3 years, M.	Blood passed; prolapse; tumour in abdomen	5 days, death	Colon descending and part of rectum into rectum.
112	Abercrombie, l. c., p. 124	4 years, M.	"It exactly resembled the previous one, except that it was not so extensive"	5 or 6 days, death	Colon descending and part of rectum into rectum.
113	Neumann, l. c. (No. 130, Pilz)	6 years, M.	Blood passed; no tumour noticed in abdomen, but one was felt in the rectum	6 days, death	Ileum, cæcum, and colon into rectum.
114	E. Mayer, Percussion der Unterleibs, p. 85 (No. 140, Pilz)	8 years, F.	Blood passed; prolapse	11 days, death	Cæcum and colon into rectum.

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
115	Thomann, Hansen, No. 47 (148, Pilz)	11 years, M.	Prolapse	Some days, death	Ileum, cæcum, and colon into colon and rectum.	
116	Dr. H. C. Rose, Med. Times and Gaz., June 8, 1861, p. 597	5 mos., F.	Vomiting; passage of blood	3 days, death	Transverse into descending colon; there were no adhesions. The invagination was about four inches long.	
117	Abercrombie, l. c., p. 124	6 mos.	Vomiting; passage of bloody mucus; tumour in left side of abdomen; injections could not be made to pass up	3 days, death	Ileum and colon into sigmoid flexure; the included parts were very dark coloured, turgid, and in some places ulcerated.	
118	Bock, Schmidt's Jahrb., 146 Bd., 1870, p. 176	10 years, M.	Symptoms of intussusception, and on 17th day tumour detected on left side of abdomen; then it disappeared, owing, as was supposed, to subcutaneous injection of morphia. On 7th day after tumour reappeared; same treatment adopted. Finally symptoms of peritonitis. As the subcutaneous injections seemed so successful at first, no other treatment was adopted the second time	43 days, death	Ileum, cæcum, and colon invaginated into descending colon; perforation above intussusception had occurred.	
119	Spaeth, Virchow's Jahresb., 1869, Bd. ii, Abth. I, p. 138	36 years, M.	After two and a half months, blood stools, and tumour felt left of abdomen; then symptoms of peritonitis. It is said that no tumour could be felt in the rectum	6 mos., death	Ileum, cæcum, and colon into colon; perforation of the colon to an extent sufficient to allow the cæcum to protrude through it.	

120	Dr. Stephen Rogers, New York Med. Record, May, 1871, p. 115	7 years, M.	Passage of bloody mucus; sickness; tumour in left side of abdomen, which changed position and became harder at times. The 2nd day, diagnosis made, and injection of air and kneading tried, but given up; then salt and water tried and continued. At first only four ounces could be thrown up; then, by evening, twelve ounces. On 3rd day sixteen ounces; and on 4th day, though bloody mucus passed, eighteen ounces, and the tumour disappeared. Morphia was given also	3 days, recovery	Experiments Dr. Rogers had made on the cadaver had shown that the ileum, artificially protruded through the ileo-cæcal valve, could be pushed back by inflation. The attempt to force fluid through the valve, so as to reduce an invagination of the small intestine higher up, succeeded in two or three experiments on the cadaver without difficulty; in others it was impossible to send the fluid past the valve until a little manipulation removed the obstruction. He discussed and advocated the propriety of opening the peritoneal cavity in extreme cases, and applying taxis directly to the bowel affected	Recovery by injection treatment.
121	Dr. Plath, Caspar's Wochenschr., 1839, p. 432	14 weeks, M.	Passage of bloody mucus; sickness; fulness on left side of abdomen more distinct on the second day	3 days, death	Ileum, cæcum, and colon into colon and rectum. The cæcum was just above the orifice of the anus; the intestine was on the point of becoming gangrenous; it could not be replaced without laying open the outer sheath. (This case occurred before No. 102, in which an anal examination was made.)	
122	Thomas, Journ. f. Kinderkrank., 1866, Bd. xlvi, p. 23	22 weeks, M.	Passage of blood; tumour felt left side of abdomen; sickness and constipation; tumour not felt per rectum; clysters were of no effect	4 days, death	Ileum, cæcum, and colon into colon and sigmoid flexure, close to rectum. On attempting to withdraw the small intestine it tore at one part; there was no peritonitis.	

No.	Reference.	Age and Sex.	Symptoms and Treatment.	Duration and Result.	Details of Autopsy or of Recovery.	Remarks.
123	Thomas, Journ. f. Kinderkrank., 1866, Bd. xlvi, p. 23	6 mos., M.	Passage of blood; sickness; on third day no tumour; on fourth day tumour felt by rectum; a sound pushed the tumour back	3 days, death	Ileum, cæcum, and colon into colon and rectum; no peritonitis. Considerable force was required to withdraw the intestine, aided by a push from below.	
124	Ditto	23 weeks, M.	Passage of blood; tumour in abdomen second day: nothing felt by rectum; clysters, insufflation, and use of sound without avail	3 days, death	Colon ascending and transverse into descending; suspected to have been partially reduced; no peritonitis; no statement as to possibility of reduction.	
125	Ditto	16 mos., M.	Tumour left side of abdomen and prolapse; the latter was pushed back; the anus remained patent: The next day patient seemed better, but still passed bloody slime, and the anus was still patent. On the following day the anus closed; the peristaltic action was not re-established, probably owing to the long constriction	5 days, death	At the post-mortem the intestine was found to have been replaced	Dr. Schütz (Prag. Vierteljahrs., 1868, Bd. ii, p. 10) insists on the value of relaxation of the anal sphincter as a sign of intussusception.
126	Judson, Southern Med. and Surg. Journal (Gaz. Med., 1837)	5 mos., M.	Sickness; passage of blood; no note as to tumour in abdomen or in rectum. Death appears to have occurred within twelve hours of the passage of the blood and about twenty-four of the first symptoms	1 day, death	Ileum, cæcum, and colon invaginated into rectum, reaching six inches below the sigmoid flexure. It is noted that there was no peritonitis, nor adhesions, nor any effusion, but that the intestine was gangrenous (no description, only black from congestion ?). An attempt to withdraw the invaginated bowel was	

					unsuccessful, and the reporter remarks that if an operation had been undertaken the intestine could not have been liberated.	
127	Herbst, Rust. Magazin, Bd. xvi, p. 105 (44 Pilz)	20 weeks	Passage of bloody mucus; tumour in abdomen	24 hours, death	Descending colon invaginated into the rectum.	
128	Forke, Untersuch. u. Beobach. über d. Ileus, &c., p. 39 (48 Pilz)	25 weeks, F.	Passage of bloody mucus	36 hours, death	Cæcum invaginated into the rectum.	
129	Wiegand, Hufeland's Journ., Aug., 1830, p. 63 (50 Pilz)	6 mos., M.	Passage of bloody slime	2 days, death	Ileum and cæcum into descending colon and sigmoid flexure	Could have been felt?
130	Krukenberg, Jahr. d. ambul. Klinik., Bd. ii, p. 38 (97 Pilz)	More than a year	Passage of bloody slime; tumour noticed in abdomen	18 days, death	Ileum, cæcum, and colon into the rectum.	
131	Roth, Wurtzb. Med. Zeit., Heft 3, 2, 1862	28 years, M.	Sudden, severe pain over symphysis; shivering and fever; then brownish, jelly-like motions. On the fifteenth day vomiting and prolapse of the rectum occurred, but was reduced. Afterwards a round, hard, ill-defined tumour was felt in the left iliac fossa, and on examination by the rectum an obstruction was met with produced by an oblong, hardish tumour with a slit-like opening (resembling the os tincæ) at its lower end	3 weeks, recovery	The treatment consisted of the use of long-continued warm baths, frequent injections of lukewarm water, and the administration of castor-oil occasionally during a portion of the time. Daily examinations were made per rectum, but it did not seem that the injections had any effect; finally, the tumour disappeared. It is noted as a case of spontaneous reposition; the attempts to effect reduction by surgical procedures being unavailing	Supposed spontaneous reposition.

A

CASE OF RENAL CALCULI.

BY

SAMUEL GEE, M.D.,

ASSISTANT-PHYSICIAN TO ST. BARTHOLOMEW'S HOSPITAL AND TO THE HOSPITAL FOR SICK CHILDREN.

(Received October 28th—Read November 25th, 1873.)

THE patient, whose history forms the subject of the following communication to the Society, was admitted into St. Bartholomew's Hospital on the 9th of July, 1873, under the care of Dr. Harris. The age of the patient was thirty-eight years. His father was gouty. He himself had suffered from gout since the age of twenty-seven years, having three or four fits each year.

From childhood he had been liable to attacks of pain in the right loin. Whether these attacks of pain were attended by other symptoms indicative of renal colic I am not able to say. Five months before his death the pain in the right loin became continual. And, about the same time, he noticed a swelling in the right half of his belly.

On admission into the hospital he looked pale and sickly. There was a small gouty deposit in the cartilage of the right ear. Both the great toes were much deformed by gouty disease, likewise some of the fingers.

The abdomen was prominent on the right side. By palpation, it was easy to feel a tumour, which corresponded in position with the visible swelling, and which occupied a large part of the right side of the belly, reaching inwards to the middle line, upwards to a little below the costal margin, and downwards nearly as low as Poupart's ligament. The upper part of this tumour was hard as stone; the lower part was soft, and gave an imperfect fluctuation; moreover, even in this part, hard spots could be reached by deep pressure.

He passed a natural quantity of urine; it was alkaline, of specific gravity 1015, and turbid with pus and prismatic crystals of phosphate of magnesia and ammonia.

Whilst in the hospital he suffered from constant pain. He died rather suddenly, on July 16th, seven days after admission. His death was not preceded by any symptoms of suppression of urine.

At the examination post mortem, the heart, lungs, liver, spleen, pancreas, alimentary canal, and supra-renal bodies were found to be quite healthy. The urinary bladder contained a pint of urine; its muscular coat was greatly hypertrophied. The urethra and ureters were natural.

The abdominal tumour felt during life was proved to be renal. The right kidney was greatly enlarged; its pelvis distended and sacculated; the true kidney tissue much altered, flattened, and spread out over the contents of the dilated pelvis. These contents were as follows (see Plate II, fig. 1):

i. A great calculus, weighing 36¼ ounces;
ii. About a thousand smaller calculi;
iii. A large quantity of calculous dust, and purulent urine.

The left kidney, also, was greatly enlarged; and its pelvis distended by a calculus which weighed 9¾ ounces (see Plate II, fig. 2). There was, besides, gritty purulent urine, but no smaller stones.

I. The great calculus in the right kidney weighed 36¼ ounces. It consisted of a main stem which corresponded

with the pelvis of the kidney. This stem supported ten calculous masses which corresponded with the dilated calyces. In separating the soft parts, two of these attached masses broke off; and the calculus in one calyx seems to have been originally unattached. The stone is semi-translucent, very hard, of great density, and dirty white in colour. That aspect of the calculus which during life was internal is beautifully crystalline, prismatic; some of the prisms being half an inch long. The external aspect is very smooth and shining. The structure of the stone is not uniform; running through the pelvic stem is a brown core, which comes to the surface in two places, where it is not coated with the white material. The dimensions of the stone are $6\frac{3}{4} \times 5 \times 3\frac{1}{2}$ inches. The accompanying drawing (Plate II) will make what I have said more clear.

II. Of the other stones found in the pelvis and calyces of the right kidney, eight were larger than a cherry-stone (see Postscript).

i. The largest was oval and flattened; weight 50 grains; its surface was smooth, and its colour light brown, except in one place near the middle, where a dark-brown nucleus, quite like that of the great calculus, came to the surface. Section of the stone rendered this dark nucleus very apparent.

ii & iii. Two calculi weighed respectively 45 and 30 grains; they consisted of a light-brown centre, covered with opaque white crystals.

iv—viii. The other five calculi weighed respectively 40, 33, 32, 20, and 19 grains. They were all smooth, flattened oval, and pale brown. Section of one of them showed it to consist of two parts, namely, a transparent centre almost colourless, and an opaque brown crust, both highly crystalline.

III. Besides these stones there were about a thousand calculi larger than a millet seed in circumference. Together they weighed nearly two ounces. They all had much the same characters; being for the most part smooth, thin, and flat, and the larger ones very much like melon seeds. Their

colour was pale brown. A few were coated with minute crystals. Seven hundred and twenty of these stones have been preserved.

IV. The stone in the left kidney was, like that in the right kidney, of very irregular shape, and consisted of a part which corresponded with the pelvis, and ten processes which corresponded with the calyces. The pelvic portion was of the size of a turkey's egg. The weight of the whole, nine ounces and three quarters. In all other respects this stone was very much like the other; a large part of its surface crystalline, but the crystals not so fine as in the greater calculus.

Concerning the chemical composition of the calculi—the white crystalline matter, which formed the bulk of the the large calculi, was found by Dr. Russell to consist of phosphate of magnesia and ammonia, nearly pure. The smaller calculi and the sand were composed of the same salt. The brown nucleus of the large stone was more complex in its nature, consisting of oxalate of lime, with traces of phosphate of magnesia and ammonia, carbonate of lime, and uric acid.

But my reason for submitting the notes of this case to the Fellows of this Society has been to put on record the great size of two of the stones. I believe that they are the largest renal calculi which have been noted, with one exception. According to Civiale,* Pohl found a stone, which weighed more than five pounds, in the kidney of a princess. Putting this case on one side, nine ounces seems to have been the maximum weight attained by any renal stone hitherto recorded. The largest of the stones which I have described is four times this weight. The specimens themselves are on the table; no doubt they will be long preserved among the curiosities of our Museum at St. Bartholomew's.

Postscript.—Since writing the above description, another stone of triple phosphate, weighing seventy grains, has been found imbedded in a crypt of the right kidney.

* Civiale, 'Traité de l'affection calculeuse,' 1838, p. 115.

DESCRIPTION OF PLATE II.

RENAL CALCULI (see p. 78).

Fig. 1. Large calculus, weighing 36¼ ounces, found in the right kidney. ⅛ diameter of real size.

Fig. 2. Left kidney, and contained calculus, weighing 9¾ ounces, found in its pelvis. ½ diameter of real size.

Fig. 1.

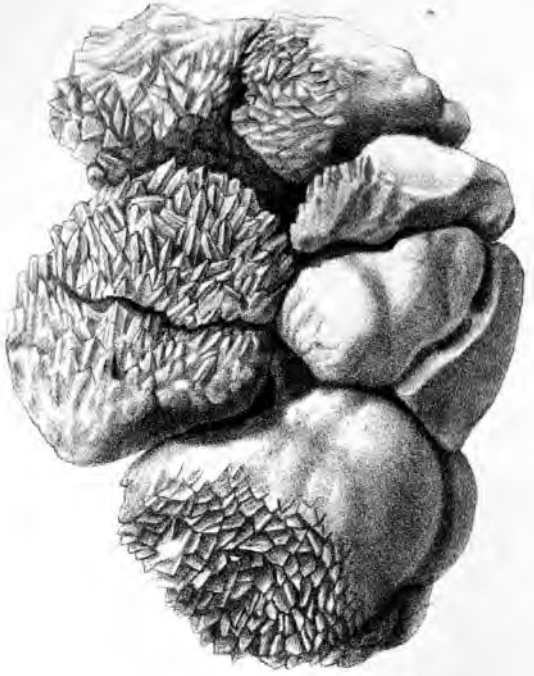
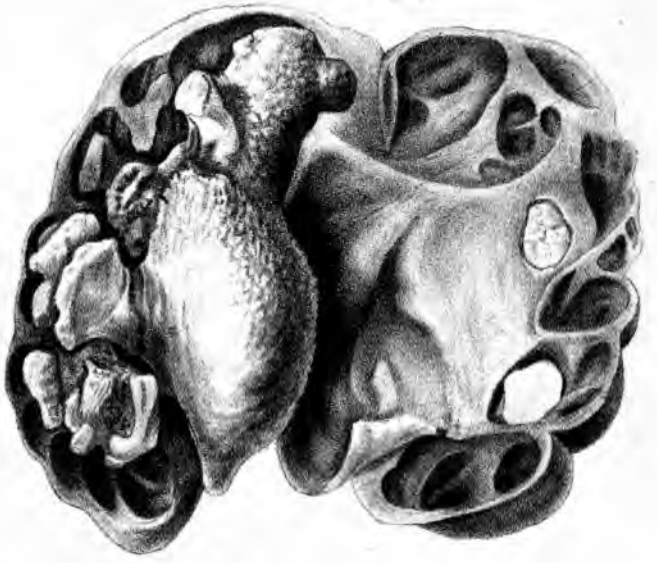


Fig. 2.



J. Godart. del. G.H. Ford lith.

Mintern Bros. imp.

THE

SUCCESSFUL TREATMENT OF ANEURISM

BY

POSITION AND RESTRICTED DIET.

BY

JOLLIFFE TUFNELL, F.R.C.S.I., M.R.I.A.,

PRESIDENT OF THE ROYAL COLLEGE OF SURGEONS IN IRELAND.

(Received November 18th—Read December 9th, 1873.)

In the paper which I published some years since upon the cure of internal aneurism by position, cases were adduced to show that consolidation of the contents of the sac of an aortic aneurism (as enunciated by Dr. Bellingham) could be systematically brought about, and not only the life of the sufferer preserved, but health with all its enjoyments restored.

That such is the fact an extended experience has confirmed; and if the plan of treatment by position be but *steadily* and *perseveringly* carried out a successful issue can (in suitable cases) almost be guaranteed. This may seem very strong language to use, considering the fatality which has hitherto attended upon internal aneurism, but I employ it advisedly, being assured of its correctness.

The question then naturally arises as to the meaning of

the term "suitable cases," and the reply is, those instances in which the aneurism springs from the front of the aorta, where the sac is entire, and the individual possesses a fibrinating power in his blood.

Case 1.—*Aneurism of the abdominal aorta cured by rest and restricted diet in thirty-seven days.*

The first case that in illustration I am about to submit to the notice of the members of the Society this evening, being accompanied by the aneurism itself, may, indeed, seem to be a contradiction of the statement; but when, in juxtaposition to the aneurism, the cause of death is exhibited, the apparent anomaly is explained; for sinking under Bright's disease, and succumbing to that fatal alternation in the structure of the kidney which characterises this affection, the cause of the death of the patient is made manifest; parenchymatous nephritis, with its interlobular cell-formation, having destroyed the circulation of the organ, perverted its normal secreting power, and thus terminated life.

The preparation and drawing are now before the members of the Society, for their inspection.

For the opportunity of observing this case I am indebted to Dr. Hugh Thomas, of Liverpool, in whose practice it occurred, and who recommended the patient to come to Dublin and place himself under my care. He arrived at the City of Dublin Hospital upon the 7th of February last, in a very exhausted condition, the steamer in which he was crossing over having been lost, and he and some of the crew saved by a life-boat; but having been landed upon a beach, they were exposed (wet through), for many hours of the night, to the full fury of the gale.

The name of the patient was John K—, his age thirty-one years, and he was a shipwright by trade.

From Dr. Thomas I learned that his father had died of cardiac disease. J. K— said that about twelve months previously, after a hard day's work, he first felt dorsal pain,

for which he consulted Dr. Thomas, who prescribed rest from labour, and the endermic use of morphine. This afforded considerable relief, and after a while he resumed his work. The pain, however, returned, and he had again to discontinue his employment. Four months afterwards aortic pulsation appeared, which gradually increased up to the time of his admission into hospital. Upon examination of the abdomen a circular aneurismal tumour was found, situated two inches below the ensiform cartilage, measuring three and a half inches in either diameter; a loud but soft bruit de sofflet accompanied the first sound of the heart, which was audible to the naked ear laid *gently* upon the surface of the abdomen; but, as usual, was lost upon the patient standing upright. The pulse was very rapid and greatly affected by position, being 104 when lying down, 120 sitting up, and 170 after walking once or twice slowly across the ward. It was intermittent also at every third beat. Sleep was interfered with in consequence of the dorsal pain, but in other respects his functions were natural.

He was left quietly in bed for a few days, in order to get over the shock consequent upon the voyage, and permitted to take such food as he had a fancy for. Upon the 17th of February treatment was commenced, and the recumbent position, with strictly regulated diet, enjoined.

The daily allowance of food was, for—

Breakfast.—2 oz. of bread and butter; 2 oz. of milk, or tea.

Dinner.—3 oz. of mutton; 3 oz. of potatoes, or bread; 4 oz. of Chancellor's claret.

Supper.—2 oz. bread and butter; 2 oz. of tea.

Total, per diem.—10 oz. of solid food and eight ounces fluid, and *no more*.

My directions were steadily maintained by the patient, who, I believe, never once exceeded or arose from the bed until permitted to do so, but continued lying constantly upon his back, or else rolling himself gently over on to one or other side.

Upon the 6th of March he reported that he *felt* the aneurism getting better.

His pulse, though still intermitting every third beat, had now fallen from 104 to an average of 69 in the minute, a dimunition of 35 beats, or of no less than 50,400 pulsations or distensions of the sac in the twenty-four hours. The dorsal pain, too, had so much declined in severity, that the injection of morphia was ordered to be discontinued, and the hydrate of chloral, in doses of thirty-five grains, administered instead. This, however, did not suffice to soothe the pain, and had also a tendency to nauseate the patient. It was therefore discontinued, and the morphia injections re-employed.

Upon the 14th of March the patient stated that he was daily becoming easier, and *that he felt he was getting well.*

He was this day examined by Mr. Timothy Holmes, who, being in Ireland, took the opportunity of visiting the patient.

On the 26th of March, K— remarked that the sensation in the aneurism was as though it was being squeezed, and upon examination by the hand it could be felt like a solid mass. The bruit, too, had all but disappeared.

Thirty-seven days had now elapsed since the treatment was commenced. He was still confined to bed. On the 14th of April he had a sharp febrile attack, and complained greatly of thirst, which was met by sucking transparent ice. The quantity of urine now passed was noticed to have very much diminished, not amounting to more than ten or twelve ounces in the twenty-four hours, and upon examination was found to be albuminous.

On the 15th of April there was a total suppression, but on the 16th the kidneys resumed their functions.

On the 21st of May he had another sharp febrile attack, accompanied by total loss of appetite and furred tongue, and his thirst was again very great.

He was now allowed, in addition to the ice (of which he took about four pounds per day), cider to drink as freely as he wished.

His pulse had gradually increased from 62 to 86.

Upon the 31st of May he was free from fever, his appetite had returned, and the quantity of urine passed had increased from twelve to sixteen ounces in the twenty-four hours.

Upon the 2nd of June there was another relapse, and a renewal of febrile symptoms, the pulse rose to 90, and the general depression was considerable. By the 13th of June the patient was sensibly emaciating. He had been sitting up for some days, but preferred the lying position. The aneurism could now be felt readily as a hard tumour to which pulsation was communicated, and could be grasped by the hand through the abdominal walls. By the 1st of July sickness and irritability of stomach set in, and became almost continuous. Towards the middle of the month he was better in some respects, but very weak. He was allowed to go out into the air and take whatever exercise he liked. By the 31st of July œdema of the feet and legs supervened, and the urine was now *loaded* with albumen.

On the 22nd of August the pupil of the right eye became dilated, whilst the left remained natural in size.

Upon the 28th of August the œdema of the feet and legs had begun sensibly to decline, whilst the urine was now literally a solution of albumen.

On the 4th of September the œdema had totally disappeared.

Upon the following morning, the 5th, after partaking of his breakfast, he said that he felt sick, and soon vomited. This was succeeded by an excruciating pain in the supra-orbital regions. Shortly after which he became quite blind, so that he could not see a sheet of white paper held before his eyes. He suffered, too, from an intense and unquenchable thirst. At 5 p.m. he became suddenly comatose, the eyes being upturned and the pupils irregular. This state continued for about ten minutes, when an improvement appeared, and as if likely to be followed by consciousness; but, instead, a violent epileptiform fit ensued, engaging all the muscles of the body, which became rigid, and the hands and feet deeply livid. The pupils were widely dilated.

Between this period and 1 a.m. of the 6th he had fourteen similar convulsions, one after another, at short intervals. Between each fit the breathing was stertorous, and the pupils contracted, dilating again directly that the convulsion came on.

He expired shortly after 1 o'clock on the morning of the 6th of September.

Sectio cadaveris.—The post-mortem examination was made thirty hours after decease.

The body was moderately nourished, and there was no appearance of œdema of the feet or legs. The surface generally was rather pale, with slight lividity of the abdominal walls.

The brain was most carefully examined. It was flabby looking on the convoluted surface, and anæmic, with some serous effusion at the base; but the cerebral and cerebellar substances, the ventricles, and every portion of the brain were perfectly healthy, and no abnormal condition could be detected, though every portion was sliced and most minutely inspected.

Upon dissecting off the pectoral muscles they were found healthy, and rather florid than otherwise, upon section.

The lungs and heart, where the sternum was raised, presented no abnormal appearance, and these organs upon removal were all found perfectly healthy; but the right pleural cavity contained a large quantity of serous effusion, the pleura itself being pale and free from any inflammatory deposit. The heart was perhaps a little larger than natural, but its valves and chambers all quite sound, and the thoracic aorta with its branches natural in every respect.

Upon reflecting the parietes of the abdomen, the peritoneum was everywhere healthy, and the viscera apparently so. The stomach was distended with air. On raising it, and turning the intestines to one side, a large purplish-coloured mass came into view, situated immediately below the pancreas, which formed the upper boundary of the tumour. It measured seven inches over its convexity. This

(the aneurism) sprang out from the anterior aspect of the aorta, immediately below the giving off of the cœliac axis, and three inches above the bifurcation. The aorta itself and its branches were all perfectly healthy, and not a sign of atheroma could be detected by the eye. The aneurism, with the kidneys attached, was then carefully removed, and the spinal column examined. It was perfect; no trace of the attachment of the tumour being perceptible, and nothing differing in appearance from that of a healthy spine from which an ordinary aorta had been detached. The liver was found fatty upon section.

The kidneys were both congested, but bore a striking difference in appearance. The right was rather smaller than natural, whilst the left was greatly enlarged, measuring five inches in length from above downwards, and three and a half inches across, with the infundibulum dilated. Upon section this organ exhibited a well-marked specimen of interstitial nephritis, with fatty degeneration of the epithelium.

The aneurism itself was *completely solid*, and upon section was found filled, from the circumference to its centre, with finely laminated fibrine. Posteriorly the aorta was healthy, with its minutest branches patulous, and performing their duty. The fibrine that filled the aneurismal sac upon either side of the spine was densely hard, whilst the aorta passed downwards in the centre somewhat triangular in form, the base of the triangle resting upon the spine, and the apex meeting the innermost of the fibrinous layers, their interspace affording a free and uninterrupted passage for the blood from the heart to the lower half of the body.

Case 2.—*Aneurism of the abdominal aorta cured by rest and restricted diet in twenty-one days.*

The next case which I shall bring under the notice of the Society is one of aneurism of the abdominal aorta cured by rest and restricted diet in twenty-one days. It occurred in the practice of Dr. Carte, physician and surgeon to the Royal

Hospital, Kilmainham, and I cannot, I think, do better than present it in Dr. Carte's own words, as contained in the following letter, addressed to myself:

"ROYAL HOSPITAL, KILMAINHAM:
28th October, 1873.

"MY DEAR SIR,—

"I send the preparation of aneurism of the abdominal aorta which I mentioned to you, and which was cured upon your plan of treatment. The patient, John R—, æt. 79, a pensioner from the 20th Dragoons, a good old soldier, who had served in India for ten years, including the Mahratta war, was admitted into the infirmary on the 4th May, 1866, suffering from aneurism of the abdominal aorta. It was situated about the bifurcation of this artery, and had a distinct bruit extending over a diameter of about $2\frac{3}{4}$ inches.

"I explained to R— the dangerous nature of the disease, and he consented readily to abide by my directions and advice, which he did. The aneurism gradually solidified, and he was allowed up at the termination of three weeks.

"From this time, and for a period of three years, he enjoyed very good health for a man of his advanced years, and he finally died upon the 28th of July, 1869, of debility and old age.

"Upon examination of the body after death I found the contents of the aneurismal sac to consist of successive layers of fibrine, disposed concentrically and very firm. Nothing could be more satisfactory than the progress and termination of this case, which contrasted strongly with another which I have had subsequently in hospital, and of which I may as well give you the history, as it will be interesting to you.

"This patient's name was William R—, a pensioner from the Royal Artillery, æt. 46. He was affected with abdominal aneurism situated above the umbilicus, and apparently measuring three inches in diameter.

"To him also I explained the true nature of his complaint, and he agreed to lie recumbent and to do all according to my directions in reference to diet, &c.; but after three days he

became discontented with the confinement to bed, and persisted in walking about the ward. As, however, the aneurism began to increase to size, and its pulsation became distressing, he again agreed to submit to treatment, but with the same result as before.

"I therefore gave up all hope of being able to treat him successfully, and the man died on the 27th of April, 1873, two months after his admission into hospital.

"J. R—, on the contrary, lived for upwards of three years after the consolidation of the aneurism, and died ultimately of debility and general break up of constitution.

"As the cases were nearly similar in character and all other respects, I have every reason to believe that an equally successful result would have taken place in W. R—'s case as in that of J. R—, had W. R— only had the patience requisite for recovery.

"The preparation which I send is very hard, from having been so long in pure spirits, but it well shows the perfect condition of the sac and the protected state of the spinal column.

"I remain,

"Very truly yours,

(Signed) "WILLIAM CARTE,

"*Physician and Surgeon, Royal Hospital, Kilmainham.*

"*To* JOLLIFFE TUFNELL, Esq."

CASE 3.—*Aneurism of the popliteal artery cured by rest and restricted diet in twelve days.*

The concluding case that I shall adduce is one of aneurism of the popliteal artery, and I believe that I am correct in saying that it is the first instance in which this disease has ever been systematically treated by position and diet alone, and that consolidation of the contents of the sac and the subsequent recovery of the patient have been
about without any interruption of the arterial st
mechanical arrest being in one way or another appli

For the notes of this case and the opportunity of observing it I am indebted to Dr. Muschamp, surgeon of the Royal Dragoons.

The patient, named Roger T—, æt. 37, was a trooper in that regiment, in which corps he had served for sixteen years. He was a steady, sober, well-conducted man, and in possession of three good-conduct badges. He had been three times in hospital for rheumatism, but never for syphilis, although the marks of suppurating bubo in the left groin showed that at some period prior to enlistment he had most probably contracted a sore that was followed by abscess. He was admitted into his regimental hospital upon the 16th of December, 1872, complaining of a sharp pain in the left knee joint, and upon examination a well-defined tumour (the size of a hen's egg) was found occupying the popliteal space. It pulsated visibly, and nearly equally in every direction. Compression of the femoral artery at once stopped the pulsation and reduced the size of the swelling. The bruit was very indistinct; in fact, could hardly be heard.

The history which the patient gave was that about a fortnight before admission he felt pain in the knee, and that some four or five days afterwards he noticed a swelling in the back of the joint. He could not account for its occurrence. Upon admission there was no excitement of the arterial system. The pulse was regular, and the heart's sound natural, but, if anything, rather feeble, and the second sound not quite as distinct as the first. The aneurism not being accompanied by any urgent symptom, there being neither heat nor fulness of the joint or other sign to indicate that the tumour sprung from the front of the artery, it was determined, in consultation, to treat the case simply by position and restricted diet.

The patient was therefore placed recumbent, with the leg raised upon a pillow and the knee slightly bent, a cold evaporating lotion being kept constantly applied *over* the joint.

Upon the 19th there was little apparent change, excepting

that the pain which had at first been described as sharp had now become rather less.

On the 22nd the pulsation was decidedly diminished, and when the hand was placed upon the tumour in the popliteal space it felt firmer.

Upon the following day the pulsation was still less distinct, and upon the 28th of December (that is, twelve days after admission into hospital) the disease was cured, the contents of the aneurismal sac being solid and devoid of pulsation.

The pulsation of the posterior tibial artery (at the ankle) had also stopped. The heat of the foot had diminished as compared with that of the opposite limb, in which the circulation was, of course, perfect.

Upon the 31st of December absorption of the fibrine filling the aneurism had commenced, and the tumour had begun to decrease in size, but for precaution sake he was kept confined to bed for a few days longer.

Upon the 15th of January he was permitted to walk about, and upon the 22nd of February was discharged from hospital, and employed at light dismounted duty, which he has ever since continued to perform.

From Dr. Muschamp I have recently heard. His letter is dated Edinburgh, October 17th, 1873. He says, "Roger T— is still in the regiment, and I am happy to tell you that he suffers no inconvenience from the obliterated aneurism; the only noticeable circumstance being that the calf of the leg is fully an inch smaller than the right, and there is still no pulsation perceptible in the posterior tibial artery at the ankle."

Before closing my remarks I would observe that but a few years since this man would have been subjected to an operation that, in the hands of the ablest surgeons of their countries—for instance, such men as Hunter, Pott, Cline, Home, Cooper, Scarpa, Blizard, Bell, Travers, Brodie, Liston, Guthrie, Dupuytren, Roux, &c.—would have terminated, *in one case out of every four*, either in the death of the patient or of his being more or less maimed for the remainder of his life.[1]

[1] *Vide* Crisp's, Norris', and Philips' Tables.

Now, how changed is the position of the patient—how altered the case! Left lying horizontally for a few days, with the affected limb at perfect rest, nature (aided by art) alone effects the cure. That similar cases to the one which I have last detailed to the Society will come before its notice, and with the same successful termination, I feel assured, for why should not a similar result be brought about in all cases of popliteal aneurism where the sac is perfect and the tumour not rapidly increasing; where the aneurism springs from the posterior aspect of the artery, protruding itself into the popliteal space (thus avoiding the joint), and in which the patient possesses a fibrinating power in his blood? These conditions happily exist in the majority of cases, the contrary being the exception to the rule, and consolidation of the contents of many a popliteal aneurism will therefore, I am convinced, be hereafter brought about simply by position alone. Why, I repeat, should they not? If aneurisms of the thoracic and abdominal aorta can be filled with fibrine in successive layers, and thus cured (cases in which the impulse that produces the destructive action is but a few inches distant from the sac), surely, with the diminished quantity of blood supplied by a smaller vessel, consolidation by deposit should, in popliteal aneurism, much more readily ensue.

LARGE ADENOCELE, COMPLICATED WITH MILK CYST.

BY

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(Received November 15th, 1873—Read January 13th, 1874.)

ELIZABETH H—, æt. 24, married, was admitted into St. Thomas's Hospital on November 27th, 1872, with a tumour of the left breast. She stated that she first noticed a swelling at the lower part of the breast, eight years ago; it was then about the size of an egg, and had gradually increased in size, but had never caused her any pain, except during the last month, when she complained of a shooting pain below the nipple. She had been married two years, and had an infant, seven months old, which she was suckling until she sought admission into the hospital. She was of spare frame, but her general health was good, and the catamenial period was reported to be regular; the bowels habitually constipated.

Three years since she went to St. Bartholomew's Hospital, and was there for two months, but refused to have any operation performed.

At the time of her admission into St. Thomas's Hospital the tumour, or rather the enlarged breast, measured twenty-

six and a half inches in circumference, the nipple being six inches from the chest-wall above. It was uniform, globular, without fluctuation, and felt doughy, like the normal breast during lactation; the cutaneous veins were enormously distended. The bulk of the tumour was below the nipple; and the great weight was sustained by the patient's left hand. There was no tenderness nor discoloration at any part; but milk could be expressed from the nipple. The axillary glands were slightly enlarged.

FIG. 1.

On December 4th the tumour was removed, crescentic incisions being made, including the nipple. The breast

proper being apparently unaffected, a considerable portion of it was left: the hæmorrhage was troublesome, but was arrested by torsion of the bleeding vessels, and the edges of the wound were brought together with wire sutures, its surface having been previously sponged with spirit and water.

The patient went on well until the eighth day, when she had an alarming rigor, accompanied by high temperature and quick pulse, and arrested secretion from the wound. The subsequent formation of abscess above the breast explained these symptoms, and after that was opened she made a good recovery, and left the hospital on January 8th.

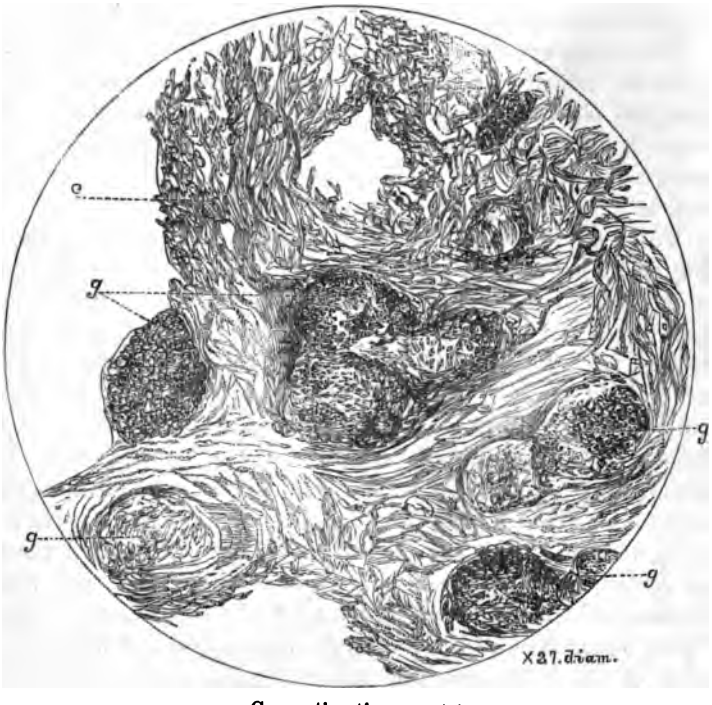
This large spheroidal tumour, the growth of which commenced, apparently, from the outer side of the breast, weighed eleven pounds, and was found, on cutting into it from behind, to consist of an enormous milk cyst, surrounded by succulent, solid walls, on which were milk tubes, and at least one extra cyst.

The walls were of different thickness at different parts; but along the section had an average density of an inch and a quarter. The greatest thickness was behind, at the outer and back part of the tumour, where the wall was two inches thick; and the thinnest part was below and to the outer side of the nipple. The structure of the walls was juicy, and, to the naked eye, resembled a section of healthy breast, with interspersed connective tissue; but it was more succulent than usual, and milk exuded freely from the open orifices of divided milk-tubes. The walls did not appear to be generally cystic, but at their thickest part a hard mass projected, as a knob, into the interior of the cyst; and this proved to be another cyst, filled with very firm cheese. The lining membrane of the large cyst was ragged and fibrinous; that of the small one was smooth. The contained milk, about two pints, was like fresh cream, and not lumpy. It was composed, according to Dr. Bernay's analysis, of the constituents of ordinary cream, with a larger proportion of albumen than usual. The solid growth weighed between eight and nine pounds.

A portion of the breast tissue which was removed was found to be continuous with the new growth; but their connection was so slight as to require the most careful examination to trace it.

Microscopic appearances.—The succulent tissue of the walls of the large cyst was composed of a large abundance of connective tissue, of acinous glandular tissue, and of ducts and vessels. The lining of the cyst consisted of the same structures, saturated with oily matter.

FIG. 2.



c. Connective tissue matrix.
g. Glandular structure.

This tumour appears to belong to the class described by Mr. Birkett as "Adenocele with ducts or sinuses, and

secretion." ('Guy's Hospital Reports' for 1855.) It commenced in a virgin breast, at or before the age of sixteen.

In reply to a recent inquiry on the subject, the patient writes that she does "not remember seeing any milk or fluid of any kind exuding from the nipple until after her confinement, and then it did not flow freely, but only now and then."

These circumstances, associated with the comparative rarity of this form of adenoid, and the unusually large size of the tumour, induce me to bring this case before the Society. When the exact nature and connections of the growth were ascertained during the operation, I regretted that an effort had not been made to save the nipple by excluding it from the section of the surface removed. This step had been carefully considered; but the size of the pendulous mass, the absence of all external trace of the original gland, and the position of the nipple seemed to forbid this attempt; and the necessary severity of the operation rendered it desirable not to protract or extend it further by removing the normal gland tissue.

Postscript.—Mrs. H— writes to me that she was confined on the 9th of November, 1873, and that her breast was "very painful for the first three or four days, and very full at the upper part towards the right breast." But at the date of the letter (between a fortnight and three weeks after delivery) this inconvenience had entirely disappeared; and she adds that the breast had ceased to be in the least degree troublesome to her.

REMARKS

ON

DISLOCATIONS

OF THE

FIRST AND SECOND PIECES OF THE STERNUM.

BY

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(Received November 11th, 1873—Read January 13th, 1874.)

The object of this paper is to offer an explanation of the mode of occurrence of displacement of the first and second pieces of the sternum, illustrated by cases coming under observation at the London Hospital, and to show that their peculiar features are dependent on the anatomy of the articulation existing between them.

It is generally admitted by surgical writers that both displacements and fractures of the sternum may result either from direct or indirect violence, and that these injuries frequently complicate fractures and dislocations of the spine. But very different, sometimes conflicting, and often indefinite explanations have been put forward in reference to their mode of occurrence. In the article on "Injuries to the Chest," in the 'System of Surgery,' edited by Mr. Holmes,

Mr. Poland has collected 14 examples of dislocation of the first and second pieces of the sternum, and of these cases the displacement was due or was attributed in 4 to force applied immediately to the sternum, in 1 to violent lateral compression of the chest, in 1 to muscular action, in 1 to flexion of the body forward as the result of a fall, in 3 to falls from a height on to the back, in 2 to falls from a height, the part struck not being mentioned, whilst in 1 the cause is not stated. Precisely similar causes have been assigned to fractures of the sternum, cases having been recorded as resulting from falls on to the back, falls on to the nates, falls on to the head, muscular action, and direct blows. In fact, the two classes of injury have so much in common that some writers include the displacements under the head of fractures of the sternum. Indeed, whether the one or the other injury occurs in any particular case would appear to depend partly on the mode of application and direction of the force, and partly on the relative strength of the articulation and of the weakest part of the bone.

The resemblance between the two classes of injury is increased by the well-established circumstance that, in the large majority of cases of both, the upper fragment lies behind the lower. In displacements, for instance, the manubrium is almost invariably found with its lower extremity concealed by the upper extremity of the gladiolus, the second pair of ribs remaining attached to the manubrium, and the strong posterior layer of periosteum untorn but stripped up from the bone holding the two segments together. The only recorded exception, so far as I know, to this disposition of the segments is related by Sabatier. A man, of about 60 years of age, was thrown from a height of thirty feet into a ditch, falling on to his back and coming in contact with a large stone. A displacement of the sternum resulted, the gladiolus being buried to the extent of 20 millimetres behind the manubrium. The patient died on the eighth day. On opening the body a large quantity of blood was found extravasated beneath the ligaments and in the substance of the lungs. In commenting on this case, M. Maisonneuve re-

marks that it would prove the possibility of a dislocation of the second piece of the sternum behind the first, from indirect violence, or, as he terms it, by contre-coup, if the extreme conciseness of the details did not leave some doubt about the mechanism of the lesion, and he avows his belief that the nature of the displacement, with the accompanying contusion of the lung, and extravasation of blood, points rather to a direct than to an indirect cause. This criticism appears to me to be reasonable and just, but until we are in possession of other instances of the lesion related more completely than the case seen by Sabatier, we should not be justified in drawing a positive conclusion on the mode in which the unusual position of the segments is produced.

At first sight it might appear that there could not be any room for controversy as to the mechanism of fractures and displacements occurring from *direct* violence, and accordingly the position of the upper segment behind the lower has been generally attributed to the direct force of the blow pushing it backwards. But M. Maisonneuve has suggested that the same effect may be produced by force applied to the body of the sternum, the elasticity of the ribs and costal cartilages attached to it causing it to rebound after it has been depressed. This suggestion appears to me to furnish a more probable explanation, because the lower and more prominent position of the gladiolus, and the greater extent of surface presented by it, render it more exposed to violence. The same effect would also be produced by pressure applied towards the lower end of the gladiolus, whereby the upper end would be tilted forward, and separation of the two segments effected. In other cases, again, the question may arise whether the lesion is to be regarded as due to direct or indirect force. Sabatier's case, which has been related above, is a case in point. The instances of fracture and displacement, which complicate fractures and dislocations of the spine, furnish other examples. Some surgeons attribute the injuries to the sternum occurring under these circumstances to a violent descent of the chin on to the manubrium, whilst others explain them either indefinitely by calling them the

effect of counterstroke, or by a doubling forward of the sternum caused by forcible flexion of the spine. It is evident that the first explanation, the descent of the chin on to the manubrium, would bring the lesions under the head of lesions caused by direct violence, and that the two latter would assign them to the category of lesions caused by indirect violence. Strict attention to the circumstances under which the accident occurs in each particular case can alone furnish the correct interpretation.

That violent descent of the chin on to the sternum is sufficient to occasion a fracture of the bone, not only seems, *à priori*, probable, but is supported by the record of cases. The following case, which occurred at the London Hospital, in June, 1871, in the practice of my colleague, Mr. Couper, who has kindly permitted me to quote it, appears to corroborate this view.

The notes of the case were supplied to me by my late house surgeon, Mr. John Cooke, who took them at the time.

Henry B—, æt. 30, carver and acrobat, a strong, muscular, and healthy man, was turning a double summersault forwards, when he fell about ten feet on to the back of his head. He came down on some tan, and his head was violently flexed on to his chest. On admission into the hospital he exhibited all the symptoms of a fracture of the spine in the cervical region, and a fracture of the sternum near the junction of the manubrium with the gladiolus. He lived thirty-six hours. At the post-mortem examination the following injuries were found: Rupture of the intervertebral substance, between the sixth and seventh cervical vertebræ; of the anterior common ligament in part; of the posterior common, the interspinous, supraspinous, capsular and yellow ligaments. The sternum was fractured obliquely from a point between the first and second costal cartilages on the right side, to the third costal cartilage on the left, the line of fracture traversing the articulation between the manubrium and the gladiolus. The obliquity of the fracture accords with the effect likely to be produced by the chin inclined to one side, and pressing down upon the sternum, and it is

obvious that disruption of the ligaments joining two of the cervical vertebræ would allow much freer motion of the head than in the normal state, or than would have been possible if the injury to the spine had been situated in the dorsal region. I call special attention to these circumstances, because I am inclined to think that the chin is much more likely to occasion a fracture than a displacement, and that it is mostly if not entirely in cases of injury to the cervical portion of the spine that such a cause may come into operation.

It is remarkable that neither Mr. Poland nor Dr. Hamilton makes any allusion to contact of the chin with the sternum as a cause of fracture or displacement. Are we to conclude from their silence that they have rejected it as an efficient cause of these lesions, or that it did not come under their notice or consideration? Possibly they may have rejected it on the ground that there were more satisfactory means of explaining the injuries attributed to it. And certainly in some of the cases thus explained, other causes, such as forcible flexion and extension of the dorsal portion of the spinal column, have been the efficient agents. In illustration of this, I may adduce a case which came under my care at the London Hospital, in January, 1871. The patient, a man thirty-seven years of age, was walking in the street when some scaffolding fell upon his head. The hat which he was wearing was completely crushed, and his scalp was torn away from his skull. The spinous processes of the third and fourth dorsal vertebræ were fractured, and the body of the third was broken and compressed. The manubrium was displaced backwards from the gladiolus. It is evident that the force applied vertically to the head produced violent flexion of the dorsal vertebræ, mainly expending itself in crushing the body of the third vertebra. At the time it was believed that the separation of the manubrium and the gladiolus was due to the chin having been driven down on to the sternum; but there was not the slightest evidence of the fact. Subsequent consideration convinced me that the displacement was the result of flexion of the

dorsal vertebræ. The body of the third dorsal vertebra being crushed, the first and second vertebræ were driven further forwards than otherwise they could have been, and the force thus transmitted to the manubrium was far more considerable than any which it could have received if the vertebra had remained entire. This force would cause the upper end of the manubrium to be tilted forwards, and the body of the sternum being acted upon at the same time through the ribs attached to it, the strain would fall on the joint uniting the two pieces together. The lever-like action of the ribs on the two pieces of the sternum being quite sufficient to cause disruption of the joint, it would be unphilosophical to assume another cause, such as violent contact of the chin with the manubrium, which was not proved and could not be proved to have been in action.

A remarkable exemplification of the view that in cases of displacement of the manubrium from the gladiolus, occurring in connection with fractures and displacements of the spine, and from *indirect* violence generally, the lever-like action of the ribs is the efficient cause of the lesion, is afforded by the following case, which was recently under my care at the London Hospital. A man, æt. 55, was standing on the floor of a warehouse, when a bag of seed falling from a height of forty feet through a trap in the floor above, alighted on his back, striking him between the shoulders. The spinous processes of the six upper dorsal vertebræ were broken off, the body of the sixth dorsal vertebra was fractured, and the first six ribs on the left side were broken near their angles. The manubrium was displaced backwards from the gladiolus and considerably depressed. As the scapulæ were uninjured, the bag must have struck the patient's back nearly in the centre, but with more force on the left than on the right side of the spine, to occasion fracture of the six highest left ribs. The patient was quite certain that his head had not been driven downwards, so as to cause his chin to strike against the sternum, nor had he fallen either forwards or sideways against any projecting body. All his injuries were due to the fall of the bag of

seed directly on to his back. On admission into the hospital, there was a marked projection of the upper end of the gladiolus, and both it and the manubrium could be felt to rise and fall with each act of inspiration and expiration. The existence of rather extensive emphysema in the neighbourhood of the injury showed that the lung had been wounded. There were none of the symptoms of fractured spine, but towards the close of life interruption to the functions of the spinal cord began to manifest itself. Death took place in less than two days after the accident, and post-mortem examination displayed the injuries described. The explanation of the displacement of the manubrium and the gladiolus in this case appears to be this. The shock of a violent blow received by the six or seven upper dorsal vertebræ was transmitted by the true ribs to the sternum, driving that bone forwards. Owing to the greater mobility and leverage of the five lower true ribs, the gladiolus was acted upon more powerfully than the manubrium, and the result was that the strain fell on the structures uniting the two segments, and that the gladiolus was pressed forwards in front of the manubrium. This driving forwards of the gladiolus either separately or more powerfully and rapidly than the manubrium, which is held firmly by the first and second pair of ribs, seems to me the correct explanation of some recorded cases, such as that mentioned by Professor Humphry, in his work on the Human Skeleton, in which a woman who fell on her back on the pavement was found to have broken her sternum; and that of David, in which the separation of the two segments of the sternum was caused by a fall from a height, the back striking in the fall against some prominent object. Such cases have been explained hitherto either erroneously, as I believe, by a supposed stretching of the sternum between the sterno-mastoid and abdominal muscles, or vaguely by the violent shock or *contre-coup*. The explanation here offered will account also for several cases of displacement occurring as the result of falls, in which the body having alighted on the feet or nates has fallen over on to the back. The lever-like action of the

ribs is likewise brought into play, in cases of compression of the chest, as well as in cases in which the force is unilateral. In the latter class, the ribs will act on the sternum with the addition of a twisting or rotatory motion, well suited to rupture the ligaments and periosteum uniting the segments. Overarching of the back by violent contraction of the muscles would occasion a corresponding overarching of the sternum, and thus give rise to a fracture or displacement. The fractures recorded by Chaussier, as occurring during the expulsive efforts of labour, were evidently due to this cause, for the patients threw back their heads, bending their bodies, and resting occasionally on their heads and their heels. In all these cases of displacement or fracture the sternum is influenced through the medium of the ribs.

The frequency with which displacement of the manubrium and gladiolus occurs, and the pathological peculiarities of the lesion, are explained with remarkable clearness by the anatomy of the articulation by which they are united. Our English anatomical text books afford us no help in the matter, for they merely state that the first two segments of the sternum are connected by a single piece of symphysial cartilage, whereas there are really two distinct descriptions of joint to be found between them. There is the amphiarthrodial joint, which is the only one mentioned in the English manuals; and there is the diarthrodial or that form of diarthrodial joint which is known as the arthrodia or gliding joint. This fact was brought under my observation whilst examining fresh specimens in the post-mortem theatre, with a view to determine the anatomical conditions which predisposed to the displacement, and as I could find no mention of it in the English works, I began to entertain a hope that the observation might possibly be original; but on referring to a French 'Treatise on Anatomy,' by M. Jamain, I found a brief epitome of some observations made more than thirty years ago by M. Maisonneuve. A subsequent perusal of the original memoir on 'Luxations of the Sternum,' published in July, 1842, in the 'Archives Générales de Médecine' (iii série, tome xiv), both convinced me that there was very little new

material to be collected on the subject, and occasioned some surprise that such scanty justice had been done in works on anatomy and surgery to M. Maisonneuve's careful and accurate observations. M. Maisonneuve, however, has himself remarked in his article, that the articulation which is the seat of the lesion under consideration expressly pointed out by the ancient anatomists had not even found a place in the most justly esteemed works on anatomy of his own day.[1]

According to M. Maisonneuve's observations, with which my own in great measure agree, the superior sternal articulation presents the two following forms :—" In a certain number of cases, the tissue between the manubrium and the gladiolus is a true fibro-cartilage, more dense and tenacious at the periphery where the fibrous tissue predominates, more thin and friable in the centre where it is reduced to a purely cartilaginous state. The articulation is thus a true amphiarthrosis, which has no movement but such as is permitted by the elasticity of the intermediate lamina. But in the other cases, in place of a uniform layer of substance adherent to both surfaces, each of the latter is clothed with a distinct lamina, adherent on one side, free on the other, having, in short, the aspect of diarthrodial cartilages. When this disposition exists the layer belonging to the second sternal piece is continued on each side without interruption on to the little facet destined for the cartilage of the second rib, whilst the layer belonging to the first piece adheres on the outside to the spur of the second costal cartilage. It results from this arrangement that the condro-sternal articulation of the superior piece is isolated from the proper sternal articulation, also that the cartilage of the second rib is much more strongly

[1] " There is a kind of articular motion between the handle and the body of the breast-bone which has been observed, not merely in very narrow-chested children, but sometimes even in adults."—' South's Otto,' 1831, p. 210. Reference is made to Beauchène, " Observation sur une sorte d'articulation contre-nature entre les deux premières piéces du Sternum," in Sedillot, ' Rec. period. de la Société de Médecine de Paris,' vol. xxxiii, p. 287; and to Heusinger, in ' Meckel's Archiv für die Physiologie,' vol. vi, p. 541, tab. 5, figs. 4 and 5. Otto adds, " In diseases of respiration I have also once found it in a man who died of asthma."

attached to the manubrium than to the gladiolus. This explains how it is that in the luxation of the first two pieces of the sternum the body always abandons the cartilage of the second rib, whilst the manubrium remains constantly united to its point." It also explains the presence, in pathological specimens, of a separate layer of cartilage covering each piece of the sternum. This condition is observable in a specimen in the London Hospital Museum (marked Gb e 5), which shows a displacement of the manubrium behind the gladiolus, the periosteum remaining entire. It was taken from the body of a man, thirty-six years of age, who fell into a dry pit forty-five feet deep, and fractured the first and second lumbar vertebræ, as well as some ribs and the sternum. About half an inch below the displacement of the manubrium there is a fracture through the gladiolus.

Union of the articular surfaces in the diarthrodial form of joint is maintained by some short and deep anterior and posterior fibres, passing between the edges of the manubrium and the gladiolus, and underlying the fibrous laminæ, which "clothe the anterior and posterior aspects of the sternum, and which serve as a periosteum at the same time that they constitute a true ligamentous coat." M. Maisonneuve has thus described the periosteal layers:—"Meckel has compared them, not without reason, to the anterior and posterior ligamentous coverings of the bodies of the vertebræ. The *anterior ligamentous coat* is formed of white fibres, which cross each other in every direction. The longitudinal fibres appear to proceed from the internal tendon of the sterno-mastoid muscles. These are the thinnest; the others, transverse, are especially marked at the level of the costal cartilages, upon which they are prolonged and inserted: in short, the aponeurotic expansions of the great pectoral muscles here throw themselves into it. From these different fibrous planes there results a sort of felt, possessing an enormous power of resistance in the transverse direction, but less in the longitudinal direction. It strongly adheres to the anterior face of the bones, and would break rather than permit their detachment by violence. The *posterior ligamentous coat* is less thick than the preceding;

its fibres are more distinct and less felted; they possess for the most part a longitudinal direction, and have but little connection with the chondro-sternal articulations."

Although the posterior coat is less thick than the anterior, yet it is commonly observed that the posterior coat remains entire, or nearly entire, in displacement of the sternum. This is due to the direction in which the force generally acts, namely, from behind forwards through the ribs and to the shape of the bone. The sternum, as M. Maisonneuve has pointed out, forms an arch with the convexity forwards, and with its highest point at or near the articulation between the manubrium and the gladiolus, and the strain falls necessarily on the anterior ligament. Moreover, the longitudinal direction of the fibres of the posterior ligament resists the force better than the transverse disposition of the anterior, and favours its simple stripping up from the bone as the gladiolus is carried forwards and upwards in front of the manubrium, and the end of the latter becomes inserted like a wedge between the periosteum and the body of the bone. This view is confirmed by the fact that when the violence is direct and proceeds from the front, the posterior layer may be torn, whilst the anterior remains entire. An interesting specimen was recently removed by my colleague, Mr. Waren Tay, from a patient at the hospital, who had fallen from a height on to the sternum. The bone was broken across, near the middle, and the ends of the fragments were bent inwards, the posterior layer of ligament being ruptured, and the anterior retaining its continuity.

The posterior layer of periosteum adheres but slightly to the bone above the level of the third pair of ribs, but below that point its connections are more intimate, and hence, as pointed out by M. Maisonneuve, it will generally be found stripped from the bone as far as, and no farther than, the third ribs.

The general anatomical conditions of the displacement which have been enumerated are exemplified in the specimen which I removed from the body of the patient, whose sternum was dislocated by the fall of a bag of seed on to the

upper part of his back. It will be seen that the second pair of ribs adheres to the manubrium, that the two segments are held together merely by a strip of the periosteum covering their posterior surfaces, and that the periosteum has not been separated farther than the level of the third pair of ribs.

The articular end, both of the manubrium and the gladiolus, had doubtless been covered each by a distinct layer of cartilage at an earlier period of life, but it would appear that these layers had been in process either of absorption or of ossification, for the ends of the bones show only traces of cartilage here and there.

There are one or two points on which my observations differ from those of M. Maisonneuve. In respect to the relative frequency of the two kinds of joint, M. Maisonneuve states that the majority of sterna exhibit the diarthrodial form. And he places the proportion as high as three out of five specimens. My own observations would give a slight preponderance to the amphiarthrodial description, reckoning adults only, for if children be included the preponderance would be much more considerable. Again, M. Maisonneuve remarked the occurrence of the diarthrodial form more often in the female than in the male, whereas I have found it more frequently in males than females. It is to be regretted that M. Maisonneuve does not state the number of the specimens which he examined anatomically. Up to the present time I have examined 100 specimens, with the following results.

Out of 100 specimens, 51 exhibited the amphiarthrodial, 32 the diarthrodial, and 11 an intermediate variety of joint, and 6 had undergone ossification. The several series were thus composed:

Amphiarthrodial.—Adult males, 26; adult females, 17; boys, 4; girls, 4=51.

Diarthrodial.—Adult males, 22; adult females, 9; boy, 1=32.

Ossified.—Adult males 4, of the respective ages of 30, 36,

53, and 65; adult females 2, of the respective ages of 73 and 76=6.

Intermediate.—Adult males, 9; adult females, 2=11.

Under the head of the intermediate variety of joint I include all the specimens in which the separation between the two pieces is incomplete. Either a small—in some cases a very small—cavity exists in the centre of the uniting tissue, or a separation is found on one side and not on the other, or a few fibres pass across the interspace, and connect the layers of cartilage. On the whole, the affinity of these specimens seems greater to the diarthrodial than to the amphiarthrodial form of joint. On the other hand, I regard the ossified specimens as belonging exclusively to the amphiarthrodial series, as it appears extremely improbable that ossific deposit would invade and destroy the interspace which exists in the diarthrodial variety.

M. Maisonneuve found the diarthrodial joint at all ages and even in very old people without a trace of ossification, and I have found it in the child as well as in old people of seventy and eighty years of age. He has made the curious observation that the diarthrodial form is found more frequently in the adult and old person than in the child, and my own observations bear this out. I am therefore inclined to believe that the diarthrodial joint is formed by absorption occurring during the period of youth, but I have not sufficient data for the settlement of the question.

Another point on which I differ from M. Maisonneuve is in respect to the part played by the clavicle in producing displacements of the sternum. M. Maisonneuve attaches great importance to the action of the clavicle. It forms, he says, a solid buttress, and in falls on to the neck and shoulders transmits the shock to the sternum from above, whilst the middle and lower ribs convey it from below. Thus pressed between two forces the sternum yields at its weakest point. He adduces the fact of the frequent fracture of the scapula and clavicle, to show that the force was transmitted along that line. I do not deny that the clavicles

may have some influence, and in some cases considerable influence, over the manubrium, but that their action is not necessary to the production of the dislocation is, I think, conclusively shown by the case which has been related, in which the luxation was caused by force applied solely to the dorsal vertebræ and the ribs. A bag of seed fell on to the six upper dorsal vertebræ without touching the scapulæ, and the shock was therefore conveyed to the manubrium by the first and second pairs of ribs. These ribs are united to the manubrium by far less movable joints than the sterno-clavicular joint, and must have a correspondingly greater power over it, not only in forcible flexion and extension of the dorsal portion of the spine, but also in cases in which the violence is applied unilaterally. If a recent specimen be examined, in which the diarthrodial joint exists, or in which there is a tolerably thick symphysial cartilage, it will be found that in addition to slight forward and backward movements (differing much in degree in different specimens), some amount of rotatory motion can be obtained. The occurrence of dislocation from unilateral violence is greatly favoured by this mobility, and I think it will be obvious that the first and second ribs must be chiefly concerned in giving the necessary twist to the manubrium.

The influence exerted by the first and second pairs of ribs over the manubrium receives illustration from the following cases, which have recently come under my observation at the London Hospital.

Case I.—Wm. C—, æt. 41, fell off a plank on to the ground. The height of the plank above the ground was about fifteen feet. He alighted on his back, and the two upper dorsal vertebræ came in contact with a piece of timber a foot square. Immediately after the accident he experienced great pain not only at the seat of injury, but also over the sternum, at the point of junction of the manubrium and the gladiolus, between the cartilages of the second ribs. On examination I could not detect any fracture of the vertebræ, or fracture or dislocation of the sternum, but there was

marked tenderness on pressure over the
point indicated, and this remained evident
In this case there had been a severe strain
force conveyed by the second ribs not having
to occasion a dislocation.

Case 2.—A man, æt. 26, admitted under my
Mr. Couper, fell off a
neck, the latter coming
The accident caused a
cervical vertebræ, with
processes. The sternum was
left side was separated from
tion the sternum was found
strong, and the joint between the
was seen to be obliterated by
peculiar conditions of the sternum,
the first rib occasioned its separation
instead of causing injury to the sternum

In conclusion, I will state the views
paper in the following propositions.

1. The frequent presence of an
the manubrium and the gladiol
displacement in preference to f
ligamentous fibres keeping the
gladiolus in contact are generally w
part of the bone. Whenever the con
fracture will occur in preference to dislocatio

2. Separation of the manubrium and the gl
occur from forcible flexion of the dorsal portion of
and is most likely to occur when one or more of th
vertebræ is crushed or fractured. Fracture may resu
the same cause.

3. Separation of the manubrium and the gladiolus
occur from forcible extension of the dorsal spine, as, fo
instance, from falls or blows on the back or muscular action.
Fracture may also occur from this cause.

4. Both in flexion and extension of the spine the sternum is acted on through the ribs, and the greater length and leverage of the lower ribs causes the gladiolus to be thrust forwards in front of the manubrium.

5. Many cases of fracture or dislocation attributed to direct muscular action or to counterstroke were due to the leverage of the ribs brought into action by violence applied to the back.

6. Fracture of the sternum, but scarcely dislocation, might be occasioned by violent descent of the chin on to the sternum, and this might occur in injuries to the *cervical* portion of the spine.

7. The anatomy of the joint between the manubrium and the gladiolus explains several circumstances noted in pathological specimens, such as the presence of a distinct layer of cartilage covering the ends of each segment, the adhesion of the second costal cartilages to the manubrium, the rupture of the anterior layer of periosteum, and the stripping up of the posterior layer of periosteum as far as the third pair of ribs.

8. The diarthrodial form of joint has been more frequently met with by myself in males than in females, and this concurs with the greater exposure of males to violence of all kinds in favouring dislocation in preference to fracture.

In illustration of the remarks made in the paper, two sterna have been prepared to show the two different forms of joint. The specimen showing the amphiarthrodial joint was taken from the body of a male; that showing the diarthrodial form, from a female. The latter specimen is interesting also from the fact that eight ribs on each side articulate separately with the sternum. The London Hospital Museum contains another specimen in which eight ribs join the sternum, and Professor Humphry mentions one in the Anatomical Museum at Cambridge. Besides these two specimens, there are some others of the upper part of the sternum, cut vertically to show the joints. One of the diarthrodial specimens was taken from an old man, seventy-seven years of age. Another

showing the amphiarthrodial form with scarcely any cartilage, no motion, and apparently commencing ossific union, from a young man of twenty-two.

The usual condition in young subjects is illustrated by a sternum with the costal cartilages and part of the ribs attached, removed from the body of a boy, eleven years of age. The specimen shows fracture of five ribs on the right side—the second, third, fourth, fifth, six, and seventh. He had been run over, and I may, perhaps, incidentally mention that the accident also caused laceration of the right lung, and a slight laceration of the left lobe of the liver close to the longitudinal fissure, with rupture of a branch of the left hepatic duct, causing extensive effusion of bile into the peritoneal cavity and peritonitis. At the post-mortem examination the coils of intestine were coated with a thick layer of lymph stained with bile. The stools passed after the accident were at first quite white, rendering the nature of the injury apparent during life.

In this specimen an indication of commencing division of the symphysial cartilage seems to exist in the form of a slight central depression.

The other specimens on the tables are the dislocated sternum of the man who was struck by a bag of seed on the back, and the specimen (marked Gb e 5) from the London Hospital Museum.

CASES

OF

DISORDERED MUSCULAR MOVEMENT

ILLUSTRATING

THE USES OF HEMLOCK.

BY

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(Received Dec. 8th, 1873, and Jan. 13th, 1874—Read Dec. 9th, 1873, and Jan. 27th, 1874.)

The four subjects of chronic muscular spasm whose medical histories are briefly given below having experienced no relief from prolonged and varied treatment, have been selected as fitting cases in which to employ hemlock and oppose its full power against the spasm, with the view of obtaining, and at the same time thoroughly testing, its remedial influence. The result, it will be seen, is encouraging.

I have availed myself of the opportunity of calling attention to the advantages which the surgeon may derive from the judicious use of a plant which relaxes muscular fibre without diminishing the common sensibility or disturbing the intellectual functions.[1]

[1] The action of hemlock on three of the patients was demonstrated at the meeting.

Coneism,[1] or the proper action of hemlock, and the degree to which it may be induced, are also incidentally considered in this communication.

CASE 1.—*Chronic intermittent spasm of the right pectoral muscles and the left sterno-mastoid, and mass of muscles on the side of the neck* (splenius, complexus, &c.)—*Spasmodic torticollis.*

The patient, W. H—, æt. 44, of dark complexion, is a strong, and otherwise healthy, muscular man, calm, intelligent, temperate, and free from constitutional disease. He is the father of eight healthy children, the eldest being twenty-one years of age. Excepting a mild attack of rheumatic fever which he experienced six years ago, the patient has been free from illness. As a millwright he was, when the present affection came on, chiefly employed in making enlarged models in wood from drawings. For the two years preceding the appearance of the spasm he was greatly overworked, and twice in the year undertook night and day work for a period of six weeks. He assigns this as the cause of the affection, for he was happy and in comfortable circumstances when it attacked him. Spasm first appeared in the arm five years ago, involved the muscles of the neck two years ago, and began to extend, as well as rotate the head, four months afterwards.

The seat and character of the spasm had never altered, and it had gradually increased in severity, notwithstanding prolonged and judicious treatment in some of our large hospitals, until natural sleep became almost impossible and life a burden. He was admitted into St. Thomas's Hospital on the 27th October, 1873. At this time the face was drawn permanently upwards and to the right; the head was still further extended and rotated in the same direction by a con-

[1] Κώνειον, hemlock.

stant succession of jerks; and the right arm was drawn across the chest and plucked still further to the left side with every movement of the head.

The neck was full and hard. It was worn bare, for he could not tolerate the restraint of a collar or neck-tie. The patient estimates the spasmodic force of the cervical muscles at fifty pounds. A forced side-glance directed downwards from a half horizontal face gave the patient a remarkably supercilious and highly characteristic expression. He had not slept without the aid of morphia for three months, but the general health was not impaired.

Treatment.—I gave him ʒj of the succus conii in the prescribing-room, and increased the dose ʒss each day until the tenth day, when he was taking ʒiij twice a day, at 11 a.m. and at 9 p.m. This was continued until the nineteenth day, and from this date to the fifty-third day, with an intermission of two days preceding his appearance at the Society, he took a single dose of ʒiij every morning.

From the fifty-third to the fifty-seventh day he took ʒiv twice a day, with an interval of twelve hours between each dose. From the fifty-seventh to the seventieth day, with an intermission of one day, he took ʒiiiss of the succus every morning. For the last week he has not taken any medicine.

Effects of the hemlock.—To speak generally, the patient and everybody about him were conscious of a decided improvement, and when his attention was diverted from himself, by reading or other mental occupation, the head was quiet. As far as I could judge, the spasm was greatly weakened, and after the forty-eighth day it became subservient to the will. He usually offered the right hand readily, and for several weeks the spasm had left the sterno-mastoid, and the face was raised upwards and to the *left* when uncontrolled by the will; but the jerk was rarely seen, the movement of the head consisting of a slow rotation or twist.

The improvement was still more marked through the night. During his stay in the hospital he never had a single dose of hypnotic, and was nightly refreshed by sound sleep.

During the action of the medicine the movements were

not diminished, but towards its decline and for some hours afterwards there was decided and often great relief. When taking only a single dose daily, at 11 a.m., he found that the most restless period was from 7 a.m. until the time of taking the dose.

Effects of the hemlock.—A fluid ounce of the succus caused some giddiness and weakness of the knees, lasting for three quarters of an hour; short of two fluid ounces there was no marked effect on the spasm. Three and a half fluid ounces caused great muscular relaxation, and at the end of two or two hours and a half, when the action had attained its maximum force, there was inability to rise from the sitting posture or to walk without assistance, and as often as the knees were flexed to a right angle or a little less he fell, and was unable to rise without assistance. There were complete ptosis and relaxation of the orbicularis, moderate dilatation of the pupils, double or multiple vision, unless he carefully rested his gaze on a near object, until the optic axes converged, and the eyes were meanwhile adapted to discern it clearly. The lower jaw had a slight tendency to hang, and the force of the masseters and temporals was barely sufficient to divide a piece of bread placed between the incisors—as the patient said: he "could not chew nor turn the food in his mouth." Deglutition was slow, painful, and imperfect, and the meal was laid aside until the end of the third or fourth hour. The effects came on a quarter of an hour after taking the medicine, and gradually increased in intensity until the end of the second or third hour, and then gradually declined and disappeared towards the end of the fifth hour. The effects were most severely felt on the left side, and remained longest in the muscles of the head. For a further account of this case see the Appendix to the paper.

Case 2.—*Intermittent spasm of the left sterno-mastoid and mass of muscles on the right side and back of the neck (spasmodic torticollis).*

The affection in this case is identical with that of the

former, less the spasm of the arm; and, like the former, the patient, John M—, æt. 40, is an industrious, temperate, and healthy man, muscularly strong, of dark complexion, and in the prime of life; weight 11 stone. He has never had any illness, and his children are healthy and free from nervous disorder. His occupation is brass-plate engraving; he works with chisel and mallet, and in order to maintain an oblique view of the plate at which he is working, the head is slightly raised and rotated to the right. He has had much domestic trouble during the last year, and has worked long hours.

He is conscious that the affection came on "with a feeling that the eyes were not quite parallel, and that he must needs twist his head farther round to the right in order to obtain an accurate view of the block." This feeling gradually increased until the head was distinctly twitched to the right.

The spasm rapidly increased and incapacitated him from work. It had existed six weeks when he came to me, at St. Thomas's Hospital, with the right hand upon the right side of the face, pushing the head round to the left. He said his arm was wearied with controlling the head, and yet he dared not withdraw it. He did so to satisfy me, and the face was immediately plucked round to the right shoulder, the head pulled backwards, and the chin forcibly tilted upwards as if he were going to fall backwards in a state of opisthotonos.

Treatment.—I at once gave him ℥j of the succus conii. It produced moderate coneism for an hour, but did not affect the spasm. I then prescribed from ʒx to ʒxij, according to the effect, twice a day; increased the dose to ℥ij on the seventh day; to ℥iiss on the tenth day; to ℥iij on the thirte[enth] continued to take this dose twice a day as before twenty-third day, when it was increased to ℥iiiss t[wice] and continued to the thirtieth day. After this, a[t] the forty-sixth day, he took ʒiv of the juice twice a 7.30 a.m. and 7.30 p.m. On the morning of this day 9th), thinking, perhaps, that it would enable him to p[ut] a better appearance at the Medical and Chirurgical Soc[iety]

he took rather more than ʒv of the succus. My directions were seven tablespoonfuls, measured with a graduated glass, but he had used a large tablespoon which held ʒv when only moderately full. He had taken eight spoonfuls on the occasion referred to. He measured the dose for me with his spoon, and I found that it was little short of ʒvj. Since this time up to the seventieth day he regularly took ʒiv of the succus twice a day, at 7 a.m. and 7 p.m.

Effects of the conium.—For the same dose they were exactly those which were experienced by the first patient, the strength and activity of the two patients being pretty equal. But the ptosis was not quite so marked in this second case, while the weakness of the lower jaw was rather more so. The tongue was slightly affected, and there was a slowness and difficulty of articulation with a little thickness of speech. Speaking required a muscular effort, which was felt about the hyoid bone. ʒiv doses increased this difficulty, and on two or three occasions some seconds elapsed before he could exert sufficient muscular power to speak, and the voice at these times was very weak and gruff. The difficulty of deglutition was also increased, so that fluids tended to return by the nostrils. These effects on the tongue and gullet, which were associated with almost complete paralysis of the limbs, lasted for half an hour.

After the large dose taken at 7 a.m., on December 9th, the effects greatly alarmed his wife; he could neither speak nor move the legs for half an hour. The effects passed off as usual. He was able to walk to the hospital (a distance of a mile and a half) at 1 p.m. At 3 p.m. there was but feeble power in the lower jaw, and the orbicularis resisted but slightly. An hour afterwards the effects had wholly passed off. At 7 p.m. he walked three miles to the Medical and Chirurgical Society, and at 8.30 p.m. took ʒiij more, with a renewal of the effects in a more moderate degree.

Result.—On the seventh day he came into the prescribing-room with the head quiet and unsupported. The continuity of the spasm was broken, and he had long intervals of rest. Before he left the room he had one slight turn of the head,

and he said it came on a little when he was tired by walking, or if he was startled, and that there was a return to the former spasm in the morning before taking the hemlock.

The improvement continued without interruption. During the last six months he has remained well, with the exception of an occasional *tendency* to the old twist of the head when overtired. He has continued work, chiefly half time, during this period, and has passed through severe mental trials resulting from a tragic occurrence in a family who occupied part of his house. The hemlock was discontinued during the fourth month.

CASE 3.—*Chronic spasm of the extensors—chiefly the deltoid and triceps—of the right arm, and of the right pectoralis major.*

The patient, George L—, æt. 19, is a healthy young man, of regular and temperate habits. His occupation is that of a watchmaker, but he is unable to do ordinary fine work on account of the unsteadiness of the arm, which becomes strongly tremulous or jerky whenever he tries to keep it steady. It has been in this condition for five years. A year ago he was treated in one of the London hospitals during three months for "cramp in the arm." Galvanism was the chief remedy employed, but no benefit resulted. He came under my care nine months ago.

Treatment.—I treated him the first six weeks with conium, ʒvj of the succus once every day, and applied a blister once for pain in the shoulder. For the next ten weeks he took $\frac{1}{24}$ gr. strychnia twice a day, with an astringent chalybeate and mineral acid, and wore belladonna plaster over the deltoid. During the following six weeks he took from 10 to 20 grains of bromide of potassium thrice a day. For the following three weeks he took the juice of æthusa cynapium and the tincture of its fruit. None of the medicines, however, made the least impression on the spasm, and I therefore determined to try the effect of large doses of

conium. I gave ℥j at first, daily, and gradually increased the dose, so that at the end of five weeks he was taking ℥iiss. After another week I gave him three ounces for a dose every day, and he has continued it up to the present time, a period of forty days.

The effects of the hemlock in this case agreed exactly with those resulting from the same doses in Cases 1 and 2. The ptosis was nearly complete, and the relaxation of the orbicularis so complete that the eyelids appeared swollen.

The results.—While he was taking the full doses he was satisfied that there was a little diminution of the spasm; but I was unable to continue the treatment for want of an efficient juice, and the arm is now as unsteady as ever.

CASE 4.—*Epilepsy and epileptic hemiplegia.*

Cordelia S—, an intelligent little girl, 4½ years old, was strong and well at two years of age, dentition was completed, and she spoke and walked well; but at this time she struck the left temple in a fall, and lay insensible for two minutes. Seven weeks afterwards she had a succession of convulsions with unconsciousness, lasting three hours. For the following three days the right arm was continuously convulsed, and for six months she was speechless. The fits recurred after an interval of five months from the first attack, and she had one every other day for two months. During the next four months she was free from fits, and suffered only from the partial hemiplegia of the right side. After this interval, in May of the present year, the fits recurred and were stronger than they were before, and during the next three months she had from two to nine fits, each lasting about two minutes, every day. They were always attended with loss of consciousness, and the palsied limbs were always most convulsed. Towards the end of this period, in July, 1873, she came under my care, at St. Thomas's. The muscular parts of the right limbs measured from an inch to an inch and a half less in circumference

than those of the left. The right arm hung powerless by her side; the protruded tongue deviated much to the left side. She was unable to walk or stand alone; the sleep was disturbed. She is a bright, intelligent child, and presents a slight scar on the left temple, which is the site of the blow.

Treatment.—I prescribed ʒij of the succus conii twice a day, increased it a week after to ʒiij, the following week to ʒiv twice a day; on the fifteenth day to ʒv, which she took twice a day for the next seven weeks. During the following three weeks she took ʒvj, for the next two months ℥j twice a day, and for another week ℥j once a day, and then, for two weeks longer, ℥j every other day.

Effects of the hemlock.—The small doses had a quieting effect, and the nocturnal restlessness disappeared. ʒv doses caused complete muscular relaxation, and she lay motionless for two hours, apparently "sleeping soundly" during the greater part of this time. As long as the lids remained open "the eyes were fixed and she seemed unconscious." As she grew stronger and recovered the use of the limbs, it was necessary to increase the dose rapidly, in order to produce inability to walk, and having become more active she was no longer observed to "sleep," but she would try to move about for a portion of the time during the action of the medicine, which was accompanied by both ptosis and squinting.

Result.—The fits rapidly declined in number and severity, and finally ceased during the fifth week of treatment, when she began to walk alone. In the ninth week she could raise the arm to the mouth, and a fortnight later above the head. Henceforward to the present time there has been uninterrupted improvement, and but for a halt on the right leg she walks firmly and well, presents her right hand on taking leave, and is regaining the use of the arm rapidly. She sleeps soundly, and her general health and strength have greatly improved. She remains free from any tendency to convulsion.

Such is the history of these cases up to the time when they were last presented to the Society. A further account of them is given in the Appendix.

General remarks on the nature and cause of the spasm in Cases 1, 2, *and* 3.

There is no doubt, in two of these, that the disordered movements were due to overwork. The nerve vesicles presiding over the affected muscles have been kept too long intent upon identical impressions; they have been maintained in an excited condition when there was need of rest, and the sustained muscular efforts have become weakened by overwork until the exaggerated nervous irritability has at last overcome the enfeebled will. It is exactly what occurs in the intellectual centres, where the same impressions may be so often repeated as to exclude change of thought and proper rest, resulting at last in a disturbance of the just balance of reason. In the direction those muscles were first taught, and subsequently compelled to go, in that direction they now continue to go automatically and in spite of the will. The first case of the kind that arrested my attention was that of a schoolmistress, who employed much of her time in ruling her pupils' copybooks, and in doing so rotated the head rhythmically, and with emphasis, from left to right. In her declining years she lost control over this movement, and the face was twisted every second to the right shoulder, unless her attention was strongly engaged in some other matter.

In the third case there is no evidence of overwork, nor must this be regarded as a necessary condition in the production of involuntary movements such as I have described. In a number of persons the nervous irritability is strong, and the will proportionately weak; and in these an over-anxious attention to execute certain movements is often sufficient to diminish their success, or even to secure their

failure. Nervous irritability in many, if not in most, cases gains strength from every succeeding embarrassment, and ultimately supplants the will. The young watchmaker furnishes, I think, an illustration of this. He is highly nervous although he does not know it, and cannot, therefore, acknowledge it. Steadiness of hand is essential to him, and I fear something has led him to mistrust his voluntary control. In other cases (4, for example) we must relieve the intelligence of any share in the disorder; not, however, forgetting that epilepsy itself is sometimes the effect of example, or even a creature of the imagination, and as such is, in both cases, presumably within the control of the will on the first uprise of emotion.

To revert to facts I would now very briefly direct your attention to the *surgical uses of hemlock.* If you examine the patients at the end of this meeting you will find complete relaxation of the whole muscular system, and the muscles of the head and face are apparently affected to a greater degree than those of the rest of the body.

The orbicularis is incapable of resistance. The movements of the eyeball are very sluggish, and there is more or less complete ptosis. The muscles of mastication and deglutition are nearly paralysed. Speech is slow and effected with exertion; the voice is gruff, from relaxation of the laryngeal muscles. Withal, the heart and breathing are normal; sensation and intelligence are perfect, and the mind is calm. The surgeon will infer from these facts the value of conium in trismus, in spasm of the orbicularis and of the gullet, and in dislocations of joints where the action of powerful muscles resists our efforts to reduce them. To the ophthalmic surgeon conium is ready to become a valuable assistant. It at once relieves, as if by magic, that powerful spasm of the orbicularis in keratitis which is caused by photophobia; and it is savage to talk of division of this

muscle as a preliminary to incisions of the eyeball. There is one operation in which it will prove a great boon, that is, the removal of artificial substances from the gullet. Accidents are occasionally happening with false teeth. It is both merciless and dangerous to attempt the removal of such bodies from the stomach or œsophagus without first removing the spasm which their presence excites, and at the same time relaxing the tube to facilitate their extraction. I will not enlarge on this topic. From the condition of general muscular relaxation produced by hemlock you will readily make your own inferences. I will, however, ask you to bear in mind one important fact, namely, that hemlock is totally destitute of anæsthesial properties, and that patients under its influence are able to help you by their efforts, and to guide you by their sensations.

Those who are unfamiliar with the action of hemlock will probably think that there is danger in using it as I have done to-night. I can say positively that there is none. The effects of hemlock are remarkably uniform, and the dose required is proportionate to the motor activity of the individual. In order to be explicit it may be desirable that I should indicate

The extent to which coneism may be carried.—I believe that the limits of safety are usually reached when deglutition is impeded, for here we trench on an involuntary act; but it may be carried thus far with perfect safety. (See Cases 1 and 2, where this effect has been induced day after day and twice a day for more than two months.) When the gullet is affected the speech is usually slow and thick, and the voice gruff from partial paralysis of the glossal and laryngeal muscles. In such cases the respiration is not appreciably affected; but when the coneism has passed off sighing is often an after effect, and a feeling of aching or oppression around the margin of the ribs, indicating a temporary depression of the respiratory function and weariness of the diaphragm. When the gullet is weakened we shall find the muscles of mastication and expression relaxed, the eyelids drooped and swollen, and the limbs incapable of supporting

the weight of the body. In women and inactive weakly persons the voluntary muscles are usually paralysed before the gullet or tongue is affected, and so they may lie incapable of movement, when the speech is free and deglutition easy. If there be any special weakness of a set of voluntary muscles, hemlock will be sure to point it out, and so it happens that in some cases the speech is affected by a dose which in others produces equal effects upon the muscles of the trunk, and yet does not affect the tongue at all. In some cases, *cæteris paribus*, ptosis and dilatation of the pupil are less marked than in others. While hemlock may therefore be used as a measure of the power of different sets of muscles, it is otherwise perfectly uniform in its action.

As to the *duration of the coneism*.—In cases of muscular spasm I have endeavoured to keep up the coneism as long as possible. The patients themselves often plead for this on account of the return of the spasm seven or eight hours after the coneism has passed off. The duration of the coneism is, of course, in proportion to the dose; the effect of an ounce of good succus in a healthy man will usually last about an hour; that of two ounces, from two to three hours; that of three ounces, from four to five hours; and that of four ounces, from seven to nine hours. Cases 1 and 2 are frequently under the influence of hemlock fourteen, and sometimes nineteen, out of the twenty-four hours.

I have been careful elsewhere ('Old Veg. Neurotics,' p. 348, and 'The Practitioner,' vol. v, p. 342) to point out that there is great variation in the strength of the succus conii, a variation which may be expected until the requirements of pharmacy are provided for by a better organisation, and the purity of drugs is secured by proper legislation. The juice used in the treatment of the above cases was most carefully selected. The doses mentioned have reference to a succus prepared according to the Pharmacopœia, but of a strength above the average of that preparation. I employed it for about nine months in the treatment of a great number of nervous disorders. A fluid ounce was sufficient to produce decided coneism in a strong man, while half an ounce was an

equivalent dose for a woman or a weakly man. The latter part of the treatment was interrupted by the exhaustion of the hospital stock of this juice; and before I obtained a supply from Mr. Buckle, who has prepared this juice for me from plants in a more advanced stage of development than those commonly employed, I was obliged to reject several samples of hemlock juice. Some had evidently been prepared from young succulent plants, and a dose of five or six ounces was needed to produce only a moderate effect; and some were inert and fraudulent preparations, of which the ordinary extract formed the basis.

APPENDIX.

THE patient, W. H— (Case 1), remained under my care for some weeks after the preceding observations on his case were concluded. I continued the conium treatment, but owing to the difficulty and delay in procuring a succus of fair power, I was obliged to discharge him after a sojourn of nearly five months in the hospital. On leaving St. Thomas's he was admitted into Guy's Hospital, under the care of Dr. Frederick Taylor, who has kindly communicated to me the following observations. It will be seen that they agree with my own.

"W. H— was admitted on April 15th, and the following day he began the conium treatment, taking only a trial dose of two drachms. The dose was increased daily; no appreciable effect being obtained until the 20th, when two ounces produced very slight tingling of the legs, with slight relief of the spasm.

"On the 21st three ounces were given, and the eyelids drooped, and the legs became very weak; the spasm of the muscles was somewhat relieved.

"From 25th April to 2nd May he took five ounces at night (7 p.m.); this produced tingling and weakness of the feet and legs, weakness across the loins, drooping of the eyelids, inability to swallow, paralysis of the arms, with semiflexion of the fingers, and impaired vision, so that objects at a distance of ten or twelve feet looked misty, and at greater distances appeared double, treble, or even multiple.

"From May 2nd five and a half ounces; and from May 9th six ounces were ordered every night.

"From the 14th to the 23rd of May he took from four to five ounces of a fresh sample of the succus, morning and evening, with the same results. About the latter date, though acknowledging the great relief to the spasm, he complained much of general mental depression and a feeling of sinking about the stomach, which he attributed to the conium.

"The effect of the drug was decidedly favorable; the spasm was relaxed during the period immediately following the use of the medicine; quiet sleep was obtained; but as the time approached for taking another dose, the spasm again became troublesome. While he was taking the five-ounce doses the sterno-mastoid and the muscles acting on the right arm became almost entirely passive, so that he was enabled voluntarily to turn his face to the left, and to write letters with his right hand.

"After taking the juice in efficient doses for five weeks I discontinued it, to watch for the return of the spasm and to form an opinion as to ultimate cure. Before the expiration of a week the twitching movements were increasing, and as he was at that time unwilling to resume the conium on account of the peculiar mental depression which it caused, I ordered the continuous current to be applied daily. From this he obtained some relief, but he had at the same time a sleeping draught of hydrate of chloral. The conium was subsequently resumed in four-ounce doses nightly, the galvanism being continued at the same time. The first dose had a rapid and beneficial effect, giving him much more ease on the following morning.

"As a summary, I think one might say:—1. The effect

of a full dose in relieving the spasm and in procuring sleep and rest is very marked and beneficial. 2. That during the continuance of the treatment the slighter movements ceased, but others were only diminished. 3. That after five weeks' use of full doses, discontinuance was followed by increase of the contractions. 4. That the great mental depression produced in this patient by the frequent use of large doses will probably prevent their being used sufficiently long to enable one to form a reliable opinion on the curability of the affection by conium."

Dr. Taylor's conclusions from this case are exactly those which I should have deduced from my own observations, excepting that I would still regard it an open question whether we should not continue the treatment, with occasional intermissions, for a much longer time—for a period less disproportionate to the entire duration of the disease than one to twelve in such chronic cases.

The use of hemlock beyond the limits which I have prescribed would, I believe, be neither useful nor safe; and I have in this case, as well as some others, found it necessary occasionally to discontinue the use of the neurotic after the patient had been under its influence day after day, and week after week, for from twelve to eighteen hours out of the twenty-four. The sinking, powerless feeling referred to the epigastrium and hypochondria is due, I am satisfied, to depression of the phrenic centres, and to a weak, sluggish action of the diaphragm resulting therefrom. The mental depression is, I believe, but the consequence of this. Defective breathing necessarily depresses the vigour of the heart; and although this may not be outwardly manifest, it undoubtedly engenders in every case a feeling of mental depression.

July 25, 1874, Case 2.—The tendency to rotation of the head when tired, especially by walking, still continues, and the patient has gladly assented to my recommendation that he should have a complete rest from work and resume the

hemlock, with the view of removing this lingering trace of his distressing affection.

— Case 3.—This patient, having for some time continued other treatment, chiefly large doses of bromide of potassium, without the least improvement, is now about to resume hemlock.

— Case 4.—The conium was gradually discontinued, and the patient has had no medicine for the last four months. She has continued perfectly well and free from any tendency to convulsion; she has not much use of the hand, but the fingers are no longer contracted, and the muscles of the right limbs are nearly as plump as those of the left. The tongue still deviates strongly to the left.

The results of treatment in the foregoing cases are, therefore, most encouraging. Inveterate forms of spasm have been selected for two reasons:—1. To illustrate the general action and uses of conium; and 2, because, if it be proved that hemlock is curative or beneficial in the worst forms of these disorders, the inference is plain that the milder forms will more surely and readily yield to its power.

I would not, however, that any one should be misled by the supposition that *all* spasmodic affections—all cases of epilepsy, for example—are benefited by hemlock. There is much to be learnt in this direction; but, speaking generally, those spasmodic affections which may be expected to yield most readily to its influence have a cranial origin.

CASE

OF

PRIMARY EXCISION OF THE ANKLE-JOINT.

WITH OBSERVATIONS.

BY

HENRY LEE, F.R.C.S.,

SURGEON TO ST. GEORGE'S HOSPITAL.

(Received December 16th, 1873—Read January 27th, 1874.)

WILLIAM C—, October 10th, residing at 42, Ormond Yard, Great Ormond Street, was admitted into St. George's Hospital on the 20th of February, 1873. A horse had run away with him and fallen upon his right leg. The lower extremities of the tibia and fibula protruded through a horizontal wound, for about two inches on the outside, and rather to the front of the situation of the right ankle-joint. The sole of the foot was turned inwards and backward. The protruding bones were perfect, with the exception of the internal malleolus, which was broken off and left attached to the internal lateral ligament. The external lateral ligament had been completely ruptured, but none of the surrounding tendons were ascertained to have been injured. Without making any attempt to reduce the dislocation, the protruding extremities of the bones were sawn off, so as to remove

the whole of the articulating surfaces. The foot was then turned still farther inward, and the articulating surface of the astragalus was removed. This was effected by a thick narrow saw and a strong pair of bone nippers, bent at an angle. The internal malleolus was left in its position, attached to the internal lateral ligament. The cut surfaces of bone were easily brought into apposition. The wound was washed with a solution of carbolic acid, and carbolized dressings were applied.

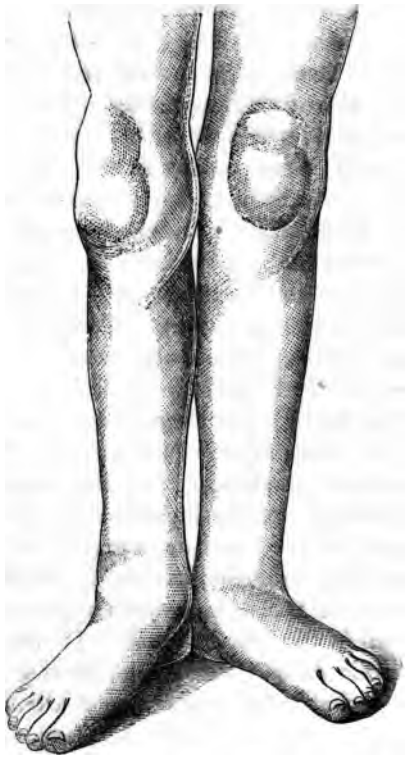
Scarcely any constitutional disturbance followed the operation. In the course of three weeks a swelling appeared on the inner side of the ankle, and an abscess formed. From this some matter continued to discharge for several days. On the 7th of April the wound through which the bones had protruded was almost healed, and the patient was able to get up.

On the 22nd of April there appeared to be good union between the lower extremity of the tibia and upper surface of the astragalus. The patient could walk fairly on the leg.

On the 3rd of May, the patient having used his leg very freely, there was considerable swelling round the ankle, and the portion of the wound which remained unhealed had a sloughy appearance. These symptoms subsided after a rest of two or three days.

May 5th.—The foot turned slightly inward and a mechanical apparatus was applied to keep it straight. He left the hospital on the 4th of June for the Atkinson Morley Convalescent Hospital, where he stayed three weeks. He then returned to St. George's for a few days. He could now walk without support and without pain. There was no swelling of the part, and the wounds were firmly healed. The astragalus appeared very strongly united to the tibia, but there was considerable motion between the bones of the tarsus. This patient was again admitted into St. George's Hospital on the 1st of October, as the stiff, high-heeled boot which he wore had chafed the ankle and produced a small ulcer on the former cicatrices on each side, and also for the purpose of

having a drawing made of the leg (see woodcut). He had walked five miles of his own accord the day before his admission, without pain or inconvenience. He was finally discharged at the end of October. The leg altogether was about half an inch shorter than the other, and he walked with a slight limp.



Mr. Hancock, in his exhaustive work on 'The Human Foot,' has given a table of thirty-seven cases in which complete resection of the ankle-joint has been performed. All these were cases of secondary amputation; and there is, I believe, no case on record, with the exception of that of which I have

now given the particulars, of *complete* primary resection of the joint.[1]

Hey (of Leeds), Gooch, and subsequently many other surgeons, have removed portions, or the whole of the protruding extremities of the tibia and fibula in cases of compound dislocation; and although many successful cases are recorded of this practice, the results on the whole have not been such as to lead to its general adoption. But, in truth, these instances were not cases of resection of the joint. The articular surfaces of the tibia and fibula were alone removed, and that often imperfectly. The cartilage covering the astragalus was left either entirely or in part. When the parts were replaced, the cut surface of the tibia would be brought into apposition with the cartilage covering the astragalus. Union between such different structures is impossible; and if the parts could unite at all, it would only be after the cartilage had become absorbed, or completely altered in its nature. Cartilage being a non-vascular tissue, undergoes changes very slowly, especially when in a comparatively healthy condition. Some time ago I amputated the leg of a girl through the knee-joint, and left the cartilage on the condyles of the femur. The girl was of a highly strumous constitution, and the wound did not heal. It became necessary, after the lapse of six or seven months, to remove a portion of the lower extremity of the femur. The cartilage covering the condyles then, to the naked eye, appeared exactly in the same condition as when the leg had been amputated in the first instance. If then, in amputation of the ankle-joint, the cartilage of the astragalus was left, it would probably be long before it would be removed. During that time no union could take place, and suppuration in the joint would necessarily be kept up.

[1] A case is recorded by Mr. Maunder, in the 'Reports of the London Hospital,' in which primary excision of the ankle-joint was performed, and in which nearly the whole of the articular cartilage on the upper surface of the astragalus was removed. The case, however, was one of a complicated nature, and the patient appears to have died of pyæmia consequent upon necrosis of a portion of the fibula.

In one of the first so-called excisions of the knee which I witnessed, the disease was confined to the femur, and the extremity of the femur alone was removed. When the parts were placed in position, the cut cancellous structure of bone was brought into contact with the smooth cartilage. The operation was not successful; and from what we now know such an operation could never have been generally introduced into surgical practice. It has indeed been suggested that union between the tibia and astragalus in excision of the ankle-joint is not desirable, but that a movable joint should, if possible, be secured.[1] Such an observation might apply to the upper extremities of the body. But even here a complete resection of a joint would be advisable where it can be performed, on account of its comparative safety. In the lower extremities firmness and stability are the conditions necessary for a useful limb.

The mode in which the tibia and fibula were dislocated in the case I have related indicate the way in which the bone may most easily be displaced in an ordinary excision of the ankle-joint, so as to facilitate the removal of the articular cartilages. In performing the operations on the dead body in imitation of the effects produced by the accident in the case related, the following steps may be taken. A longitudinal incision may be made over the internal malleolus, and as much of the periosteum as may be, and the ligaments, detached. The malleolus is then held by a pair of bone forceps and cut with a Hey's saw on a level with the ankle-joint. The saw should be broad, so as to allow this part of the operation to be completed by a pair of strong bone nippers. If the saw is narrow, the bone nippers will not pass readily into the groove.

The internal malleolus being removed, the foot is placed on its inner side, and an incision is made for two inches along the posterior border of the fibula, and is carried forward nearly at right angles from the lowest point of the external malleolus to the outer edge of the long extensor of the

[1] Garraway, 'British Medical Journal,' 1862.

toes. The periosteum and ligaments are then detached from the external malleolus, and it is removed in the same way as the internal. The lower extremity of the tibia may now be made to protrude through the wound on the outer side, and removed with the greatest facility. This accomplished, the sole of the foot is turned inward, and the upper surface of the astragalus is presented at the wound. The foot now should be held firmly, and with a thick keyhole saw the upper surface of the astragalus may be sufficiently detached without the saw interfering with the surrounding parts, to enable the surgeon to complete the operation with the cutting pliers. The whole of the cartilage on the upper part of the astragalus may in this way be removed, and a perfectly smooth surface of bone left. This is placed in apposition to the corresponding surface of the tibia, and maintained in that position. In this operation it is essential that the internal malleolus be removed or entirely detached from the tibia. The mere division of the ligaments is not sufficient. If the internal malleolus is left connected with the tibia, it prevents this bone from being dislocated outwards. After the internal malleolus is detached, the tibia and fibula may easily be dislocated outward, but when the external malleolus is detached, these bones cannot be dislocated inwards. This is illustrated in a case of Mr. Moore's, published by Mr. Hancock.[1] Mr. Moore cut down on the fibula, and excised the lower inch and a quarter of that bone. Continuing the incision in a curve across the instep to the interior point of the inner malleolus, but not through the anterior tendons, he endeavoured to dislocate the foot inwards, but could not do so.

It is evident then, as indeed may be readily proved upon the dead body, that there is a great difficulty in dislocating the tibia and fibula outward while the internal malleolus remains attached; and there is also a great difficulty in dislocating the bones of the leg inward, even when the external malleolus is removed. This also is proved by a case recorded by Mr. Hancock,[2] in which Mr. Hussey performed the following operation:

[1] 'The Human Foot,' p. 309. [2] Ib., p. 287.

"A similar incision was made over the front of the joint from behind and above the outer malleolus, to a point a little above the inner malleolus, dividing the skin and subcutaneous fat and areolar tissue only. The flap of skin was raised, the fascia opened on each side of the fibula, the peronei muscles separated from the fibula, and the bone divided by cutting pliers about two inches above the extremity. The fragment of the fibula was seized by strong forceps and drawn forwards, the ligaments were divided, and the bone removed. After dividing the ligaments at the inner side of the joint, the foot could not be everted sufficiently to throw the inner malleolus out of the wound so as to use the saw for its removal. Mr. Hussey, therefore, applied the cutting pliers to the lower end of the tibia above the malleolus. The blades were not equal in length to the whole thickness of the bone, the gouge was therefore used in cutting away so much of the cancellous structure and articular surface as seemed necessary. With his finger in the joint he felt the articular surface of the astragalus to be rough and bare of all cartilage, and he cut it away with a gouge."

I have verified the difficulties of performing both Mr. Moore's and Mr. Hussey's operations on the dead subject, but I prefer giving such illustrations from cases in which the operation has actually been performed on the living body. It would appear, then, that excision of the ankle-joint cannot be readily performed either by attempting to dislocate the bone of the leg inward, nor by trying to dislocate them outward, unless the internal malleolus be previously removed.

The mode of excising the ankle-joint which I have described, namely, by removing the internal malleolus first, and by dislocating the tibia and fibula outward, has not hitherto been described. I believe it, however, to be by far the simplest and best of all the operations of the kind. In Mr. Hancock's mode of performing excision of the ankle-joint, he commences his "incision behind, and about two inches above the external malleolus, carrying it forwards beneath that process across the front of the joint, and terminating about two inches above and behind the internal malleo-

lus."[1] In this operation the tendons of the tibialis pasticus and flexor communis have to be detached from the groove behind the internal malleolus, and if this is not done completely there is a difficulty in everting the foot so as to complete the operation.[2]

In the last edition of 'Pirrie's Surgery' it is stated—

"The operation can be most satisfactorily performed by making a semilunar incision on the outer side, raising up the flap, cutting off the under extremity of the fibula, opening the joint, bending the foot inwards, and cutting the bones with pliers."[3] In 'Holmes' Surgery' Mr. Hancock's operation is advocated. Mr. Buchanan operated by a simple incision on the outside of the ankle. The elder Moreau made an incision on each side of the ankle, and divided the tibia *in sitû*.

In Hancock's and Holmes' operation the tibia, as far as may be, is dislocated inward. In Moreau's, Pirrie's, and Buchanan's the tibia and fibula would be dislocated outward with the greatest difficulty, if at all, as no mention is made in them of the removal of the internal malleolus. The removal of the articular surfaces of the bones completely by any of these operations necessarily involves great disturbance of the surrounding parts, from the saw playing between the tendons and the bones; or if the operation be performed with a gouge, a very irregular surface of bone is left. In either case it would be extremely difficult, as experience has proved, to leave a smooth surface of the tibia, and a corresponding surface of the astragalus which would come into fair apposition; and the suppuration which would in either case probably follow would add to the danger of the patient, and retard the healing of the wound. In the case recorded at the commencement of this paper I left the internal malleolus detached, being unwilling at the time to add to the extent of the wound; but in an ordinary excision of the ankle, primary or secondary, I should prefer to remove the internal malleolus, whether detached or not.

[1] Op. cit., p. 277. [2] Ib., p. 292. [3] 3rd ed., p. 818.

ON THE TREATMENT

OF

RICKETY DEFORMITIES OF THE LEGS BY OPERATION.

BY

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(Received January 13th—Read February 10th, 1874.)

Among the cases brought in the course of the year to the Children's Hospital are a large number of rickety deformities of the legs. The majority of these are easily dealt with. If cod-liver oil is given the deformity disappears as the bones grow; or, if not, it can be corrected by some simple form of splint. But a few cases remain in which treatment even with the aid of expensive mechanical appliances is rendered difficult, and sometimes hopeless, either by the extreme degree of curvature which has been developed, or by the occurrence of those remarkable changes which the bones undergo when the acute stage of rickets is past, and which make them much stronger and more rigid than they are in their natural state. In these otherwise intractable cases I have performed an operation for dividing the bones. Two methods have been adopted. In three patients, in whom the legs were curved

outwards, the tibia was so far divided with a fine saw that I could, by bending, fracture it. In a fourth, in whom the curvature was almost directly forwards, a wedge of bone was removed from the tibia, and the fibula was cut in the right leg, and broken in the left.

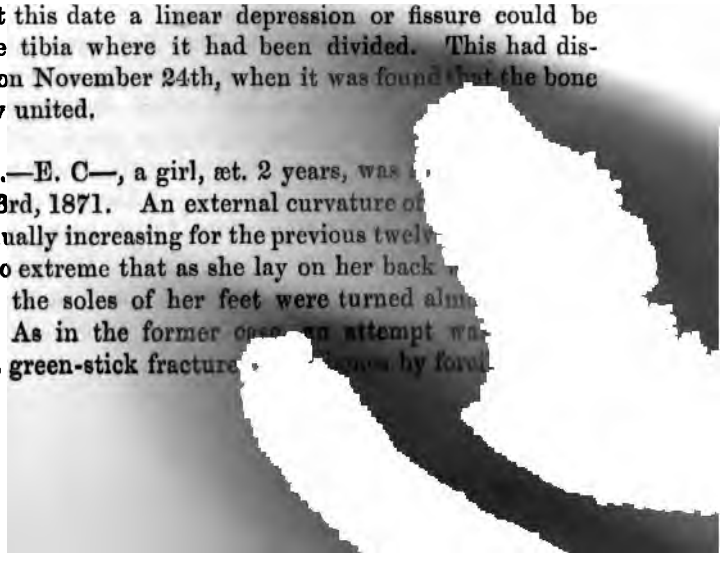
[The patients were in the next room, so that the result of the different operations could be seen.]

The following is a short account of each case, taken from the notes of Mr. Butlin, who was Registrar at the Children's Hospital when they were under treatment there.

CASE 1.—A. H—, a girl, æt. 4½, was admitted on July 1st, 1871. Curvature of the legs had begun two and a half years previously. She had been out-patient for a year and a half at the Orthopædic Hospital, where she was ordered to wear short splints extending from the inner side of the knee to the sole. Both legs were much deformed by external curvature. The deformity was said to be still increasing. On July 18th, before using the saw, I attempted to produce, as I have in some cases, a "green-stick" fracture by forcibly bending the leg; but though the tibia appeared to yield it would not snap. Having chosen the point at which the concavity of the curve was greatest, I passed a tendon-knife down to the subcutaneous surface of the tibia, and divided the periosteum transversely. The knife was then withdrawn, and a fine saw introduced, with which, by a series of very short to-and-fro movements, the bone was so far divided that I was able, by a somewhat suddenly applied force, to snap it across. There was some difficulty in getting the leg quite straight, but this was overcome when the fibula had been broken, and the tendo Achillis cut. The limb was then placed in good position on a back splint, and swung beneath a cradle, and side splints were applied. In the next few hours some venous oozing took place, and formed a puffy swelling under the skin, but this was not so great as to call for interference. The child passed a restless night, and was sometimes in a good deal of pain. On July 19th, the leg was not much swollen. The evening temperature was 101·6°. July 20th,

the patient had slept well after taking five drops of tincture of opium. The temperature both morning and evening was 100·6°. For the next few days the case went on well, there was little pain, and the temperature, which never rose above 99·6°, was on July 26th down to 98°. On August 3rd the leg was red and swollen, and fluctuation having been detected, an incision was made, which let out a small quantity of healthy pus from the neighbourhood of the operation wound. On August 21st swelling had disappeared, and on August 28th the wound was soundly healed, and the limb was put up in plaster of Paris. A few days later the child was sent to the Highgate Convalescent Hospital. She returned on September 28th. The tibia was found to be straight, and firmly united; the wound was quite sound; and the leg considerably longer than its fellow.

On October 4th the opposite tibia was divided by a similar operation, except that I bent the fibula without breaking it; and found it unnecessary to divide the tendo Achillis. The child passed a very good night after the operation, and next day her temperature in the morning was 98·2°, in the evening 100°. On October 6th she had an attack of diarrhœa, and the temperature rose to 102°. From 6th to 12th the temperature was never higher than 99·4°, and the leg was doing well. On October 12th the splints were readjusted. Nothing further occurred worthy of note, except the gradual closing of the wound, and this was complete by November 12th. At this date a linear depression or fissure could be felt in the tibia where it had been divided. This had disappeared on November 24th, when it was found that the bone was firmly united.



Case 2.—E. C—, a girl, æt. 2 years, was
vember 23rd, 1871. An external curvature o
been gradually increasing for the previous twel
was now so extreme that as she lay on her back
extended, the soles of her feet were turned alm
inwards. As in the former
produce a green-stick fracture

ing them; but they would not give way. On December 2nd the right tibia was partially sawn through, and then fractured; and the fibula was broken. The tendo Achillis was not divided. After fracturing the tibia I did not attempt to straighten the leg, but placed it, still curved, upon the splint, for it seemed important to learn whether the gradual straightening of the limb by carefully regulated pressure with side splints was not preferable to the forcible bending at the time of operation. I found, however, in this case much more difficulty in the subsequent moulding of the leg than I expected, and came to the conclusion that it is better to straighten the limb at once. On December 4th the child had suffered little or no pain until the previous evening, when she complained a good deal, but was soon relieved by a few drops of laudanum. On December 9th there was very little discharge from the wound, and scarcely any swelling of the leg. On December 11th the child disarranged her dressings, and drew her leg out of the splints. These were readjusted, and no mischief followed this disturbance of the limb. On February 2nd the note states that there had been no discharge from the wound for some time. The bone was firmly united, though not quite straight. On February 28th the back splint was removed, and simple side splints were employed.

On April 15th a similar operation was performed on the left leg, but the limb was forcibly straightened at the time. The fibula yielded without breaking.

On the 25th the note says there had been no discharge from the wound, and scarcely any swelling of the leg.

May 20th.—The wound had never discharged, and was now soundly healed. The leg was in good position. The right leg, which had been treated with firmly applied side splints had become quite straight. After the second operation on E. C— the temperature, taken every morning and evening, was never found above 99·5°. In the evening of the day after the operation it was 99°, and in the following week it rose above this point (to 99·5°) only on one occasion.

CASE 3.—A. L—, a boy, æt. 3½, was admitted on November 29th, 1871. He was healthy looking but very rickety, and subject to prolapsus ani. The curvature of the legs was not simply external, as in the previous cases, but also to some extent anterior.

On December 2nd the right tibia was cut about half through and then fractured, and the fibula forcibly straightened. The leg when placed upon the splint was still rather curved forwards; though much less so than before the operation, and I could not get it quite straight.

On December 15th a small abscess on the inner side of the leg in the subcutaneous fat behind the posterior border of the tibia was opened. This prevented for some days the satisfactory application of the side splints.

On January 10th the operation wound was closed, but the abscess was still discharging.

On February 26th the boy was found to be desquamating freely, and as he had complained of sore throat it is probable he had passed through a mild attack of scarlet fever, although no rash had been seen.

On March 14th, as the fracture was firmly united, the child was sent to Highgate.

On June 29th the left leg was treated in a similar manner, and when the limb was placed upon the splint the external curvature was seen to be almost entirely removed; but I found that, as in the opposite limb, I could not correct the anterior curve.

On July 6th the child had had no unfavorable symptom, the temperature had never risen above 100°, and was generally below 99°; but very little discharge had occurred, and this, along with all swelling, had now ceased. From this date there was nothing to notice, and he was sent to Highgate on August 15th, with his leg in very good position.

Here, with the permission of the Society, I will offer a very few remarks on these three cases, which form a group by themselves, before I relate the fourth case, in which a much more severe operation was performed. I wish to state in the

clearest terms my belief that in English practice there are very few cases of rickety curvature of the legs in which an operation is either necessary or justifiable. Those just related, however, could not, I think, have been successfully treated in any other way. Although I was not aware of it at the time, I afterwards learnt that Professor Billroth had operated in a similar case, with extremely good result, in 1861; but instead of using a saw he partially divided the bone with a fine chisel. The principle is the same; but I think, remembering the ease with which it can be performed, the operation with the saw must be the simpler, and probably also the safer method. In Billroth's patient, a little girl, aged three years, the wound, as he particularly mentions,[1] healed without suppuration.

Mr. Champneys (Radcliffe Travelling Fellow) informs me that while he was at Vienna last summer he saw Billroth repeat his operation on several occasions with very good result. Some might consider it unjustifiable to produce a compound fracture of the leg merely to remedy a deformity. But it must be borne in mind that these children were not only deformed but crippled too; they could travel but slowly and with great fatigue, and were very liable to lose their balance and fall; and the two girls as well as the boy were thus debarred from many occupations that would otherwise have been open to them as they grew up. The three cases present a series of six operations. In no instance was any serious symptom observed. In one the child had intercurrent scarlet fever, which raised his temperature and delayed his convalescence. After the remaining five operations convalescence was complete within six weeks; the temperature was never above 100°. In one abscess formed, but very soon healed. I venture to think it unnecessary to discuss the probability of non-union of fractures thus artificially produced. In all these cases union occurred quickly; and in cases of fracture in children resulting from accident, non-union, although it has been met with, is extremely rare.

[1] 'Archiv für Klin. Chir. von Langenbeck,' vol. ii, p. 657.

CASE 4.—John B—, æt. 7, was admitted on February 20th, 1871. He was very rickety in all parts of the skeleton. Both legs were curved almost directly forwards at the junction of the middle with the lower third to such an extreme degree that he could walk only a few steps at a time, and this with great difficulty. In standing or walking he lost so much of his height that he looked no taller than an ordinary child four years of age (see Plate III, fig. 1). The acute stage of rickets had passed by, and the curvature of the bones was not increasing; but although this was the case, both his deformity and his incapacity for walking were constantly aggravated by the yielding of the ligaments of his ankle-joints under the enormously increased strain to which, in the altered relation of parts, they had become subject.

On April 8th a longitudinal incision was made over the front of the tibia, where the curve was most prominent, and the periosteum was divided and carefully raised. A chain saw was then passed round the bone, and a wedge-shaped piece sawn out. The fibula was fractured and the tendo Achillis cut. The periosteum was then laid down again over the ends of the divided tibia, and fastened with a horse-hair suture, and the limb, which was brought into very good position, was placed on a back splint and swung beneath a cradle. The edges of the wound of the soft parts were loosely brought together with a silver suture. In passing the saw round the tibia, care was taken to keep it in close contact with the surface of the bone, so as to avoid injury of the neighbouring vessels and nerves. There was some difficulty in working the chain saw, as it was unavoidably doubled upon itself in the form of a narrow loop. Hæmorrhage was slight, and was seen to come chiefly from a large artery in the interior of the bone.

9th.—The child had passed a fairly good night; the temperature in the morning was 100·4°.

10th.—Doing well. Some bleeding took place to-day from the artery in the bone, but this was easily stopped by a small plug of lint soaked in a solution of perchloride of iron.

12th.—Appetite good; temperature in the morning

100·2°, in the evening 101°. The boy suffers a good deal of pain at times. The lint is to be removed from the wound, and a poultice applied.

21st.—There is great swelling on the outer aspect of the leg at the seat of the operation, and as this is evidently due to a collection of pus, an opening is to be made, and a small drainage tube inserted.

May 15th.—The splint was changed to-day. The wound looks healthy, and there is now but little discharge. No union of the bones has yet taken place.

29th.—Union of the tibia is progressing, but is not yet firm.

June 15th.—A thin plate of bone was removed this morning, consisting of an exfoliation of the whole of the cut surface of the lower piece of the tibia.

22nd.—A sequestrum with a second smaller fragment was removed from the upper piece of the tibia to-day.

July 5th.—The wound is nearly healed. The limb is very nearly straight, and the boy can easily lift it from the bed. He is to go to the Convalescent Hospital, at Highgate.

He returned on September 28th. Union of the tibia was perfectly firm, and the leg was straight, and more than an inch and a half longer than its fellow.

On October 4th the operation was repeated on the opposite leg; but in addition to the longitudinal incision a short transverse cut was made through both the integuments and the periosteum. When the chain saw was passed round the tibia rather sharp bleeding, apparently arterial, occurred, but this stopped spontaneously when the bone had been divided, and both the tibial arteries were felt beating at the ankle. As the fibula could not be broken by a moderate force it was divided with cutting bone forceps.

5th.—The boy was doing well. His temperature in the morning was 99·6°; in the evening 101·6°. He was, however, restless, and complained a good deal of pain, but he was soon easier after the bandages above the wound had been loosened.

6th.—The sutures were removed from the wound last

night, and a poultice was ordered. He was now in less pain. An erythematous blush extended from the wound down to the foot. He had passed a very good night.

On the 9th the blush had disappeared, and the wound was looking healthy. The periosteum had retracted from the ends of the bone, and the lower fragment was somewhat tilted forward.

10th.—Chloroform was given, and the ends of the bone were placed again in apposition.

24th.—An abscess behind the tibia was opened, and a small drainage tube introduced.

November 18th.—The wound had in great part healed, but there was no union of the bones. The boy's health was excellent.

December 30th.—Two small pieces of dead bone were removed from the cut ends of the tibia.

On February 18th another small piece came away.

On April 3rd the wound was almost entirely closed; the leg was straight, and the union of the tibia nearly complete. He was soon after this sent to Highgate.

When he returned he could walk well, and was in excellent general health (see Plate III, fig. 2).

The operation performed in this case may be compared to a compound fracture with a large wound. But, if such an expression may be allowed, it was a compound fracture without complication. There was no comminution of the bone, no injury of the periosteum beyond a clean cut, and no laceration of either muscles, nerves, or vessels. The operation was one, no doubt, of great severity; but, I think, under the circumstances in which the boy was placed, it was quite justifiable. It was followed by no dangerous symptoms, and it had the effect of lengthening the legs by a little more than an inch and a half, and of enabling the boy to walk without impediment. Before it was performed there was considerable distortion of the thigh bones, and it seems interesting to remark that during his prolonged confinement to bed and afterwards, as he walked on straight instead of crooked legs, this deformity was to a great extent corrected.

DESCRIPTION OF PLATE III.

Treatment of Rickety Deformities of the Legs by Operation.

CASE 4.—John B— (see page 151), in which a wedge of bone was removed from the tibia.

Fig. 1. Front view before operation.
Fig. 2. Front view after operation.

Plate III.

G. H. Ford.

Mintern Bros. imp.

CASES

OF

(SO-CALLED)

ICHTHYOSIS LINGUÆ.

BY

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(Received February 10th—Read February 24th, 1874.)

The application of the term *ichthyosis* to a morbid condition of the tongue is of quite recent date. It was Mr. Hulke who first used the name provisionally in describing a case which he brought under the notice of this Society in 1864, and which was subsequently published in the second volume of the 'Clinical Society's Reports.'

The characters of the complaint are sufficiently distinct and sufficiently well marked to warrant us in giving it a distinguishing name, and in claiming for it a separate place in the category of disease. But it may yet be doubted whether *ichthyosis* is the best term for this morbid condition. At any rate, I have preferred to entitle this paper "Cases of (so-called) Ichthyosis Linguæ," in order to show that I regard the name as still provisional, and possibly subject to alteration.

In its earlier stages the disease has much in common with simple warts, and it might be called a papilloma. It is also

in some respects similar to corns, but clinically it is widely distinguished from both these morbid conditions. Perhaps it is most akin to those rare flax-like growths which are occasionally seen on the gums, and which have been described by Mr. James Salter under the name of "Papillary Tumours of the Gum."[1]

But what are the distinguishing characters which entitle *ichthyosis linguæ* to be regarded as a separate and substantive disease? To this I reply—They are partly pathological and partly clinical.

1. It affects only the tongue and the inside of the mouth; generally it is confined, at least at its commencement, to the dorsum of the tongue. In rare instances a somewhat similar condition may be seen in other parts of the body as a congenital defect. But as a morbid state supervening upon a previously healthy tissue it is, I believe, unknown elsewhere. No other mucous membrane is subject to such an affection.

2. It slowly spreads, or, having reached a certain point, it remains stationary; but gives no pain; only some slight inconvenience. In this state it may continue for many years —twenty or thirty years—but sooner or later it becomes epitheliomatous. This, as far as I know, is a peculiar

[1] 'Guy's Hospital Reports,' 1866. The disease here referred to was first described by Sir William Fergusson, from a case which occurred in his practice at King's College Hospital, when the writer was house-surgeon of that institution. The patient was an old man, aged 80, and the disease affected the lower jaw on the right side. A portion of the gum was covered by a soft, white growth, which "looked like vegetable matter, or greatly enlarged papillæ" ('Lancet,' September 6, 1862). It was removed and carefully examined by Mr. James Salter. He has described it as "a curious white mass, consisting of coarse detached fibres, pointed and free at one extremity and attached at the other; in fact, it was a mass of papillæ, many of them nearly an inch long, similar in shape to the 'filiform' papillæ of the tongue; their surface was shreddy and broken. Among these elongated processes were a few eminences like 'fungiform' papillæ, and these had a smooth unbroken surface. In microscopical structure I found [says Mr. S.] the mass to consist almost entirely of epithelium, principally squamous and flattened, but in other parts the cells were aggregated together in groups, reminding one of the 'bird's-nest' arrangement of epithelial cancer."

feature, and certainly it is very noteworthy. The congenital cases of a somewhat similar kind, which I have already mentioned as occurring in other parts of the body, have not, to my knowledge, shown any disposition to assume the characters of epithelial cancer.

Ichthyosis linguæ manifests itself in an overgrowth of the papillary and epithelial elements of the mucous membrane, and these become white and sodden from continued immersion in the fluids of the mouth. It is the dorsum of the tongue and the filiform papillæ which are affected in the majority of instances. Indeed, it would appear that some of the special features of the disease depend upon the peculiarly bold development of the papillæ in this situation, for it never seems to spread farther back than the line of the circumvallate papillæ. Occasionally the mucosa of the gums or of the cheeks is affected in a somewhat similar manner, but generally, as I have said, it is the dorsum of the tongue which is the original and the chief seat of the disease. Sometimes the papillæ, though enormously enlarged and overloaded with epithelium, retain their separate form; at other times they are welded together into masses, and it not unfrequently happens that the same tongue presents specimens of both the discrete and the concrete arrangement. In some cases the enlarged papillæ may be seen sprouting up in small groups, very suggestive of a commencing epithelioma; in others, on the contrary, the whole of the affected surface is smooth, hard, and almost cartilaginous. The ichthyotic coating presents a silvery or a snow-white appearance, quite different from any fur that ever covers the tongue as the result of its ordinary functional changes. When the disease has once manifested itself it is very persistent. Though it sometimes responds a little to treatment, and though it varies slightly according to the state of the patient's general health, it never wholly leaves a spot which it has once attacked, and it is never cured.

The essential nature of the disease appears to be that of a chronic inflammation, accompanied by an overgrowth of the papillæ and a loss of power to throw off the effete epithe-

lium. The irritation which gives rise to this inflammation sometimes acts on the periphery of the nerves; sometimes it is situated between the periphery and the centre. In one of the cases about to be related (Case 4) the disease seemed to be due to inflammation of the inner ear, and the irritation was, no doubt, propagated along the *chorda tympani*. This is the only case I have to relate in which the irritation was remote from the affected surface. In four of the others it was peripheral. In one of these (Case 2) it was due to superficial syphilitic ulceration of wide extent and of long standing. In another (Case 7) the patient attributed it to smoking short pipes and taking the smoke into his mouth as hot as he could bear it. In a third (Case 1) smoking very strong cigars is given as the predisposing cause; while in a fourth (Case 8) the patient assigned the practice of tongue-scraping, which he seemed to carry to excess, as the only cause he could suggest. In a case noted in the Appendix (Case 5) the disease is said to have commenced in a spot upon which a tobacco-pipe had often rested.

If these sources of irritation are to be taken as the starting-points of the disease, we must assume that the patients had a strong inborn tendency to the development of warty growths under slight causes.

It is worthy of notice that in two of the cases of which I am able to exhibit drawings (Cases 3 and 4) the symmetrical arrangement of the ichthyotic patches and their peculiar form suggests an association with the lingual (gustatory) branch of the fifth pair of nerves. In one of these cases (Case 3) the disease followed rheumatic fever; the other (Case 4) is the case already alluded to in which the ichthyosis supervened upon inflammation of the inner ears. In the first of these two examples it is difficult to see any immediate connection with the fifth pair of nerves, but in the second a direct line of communication would be formed by the *chorda tympani*.

If a portion of the ichthyotic covering be examined under the microscope the appearances are very remarkable. The microscopical preparations upon which the following account

is founded were made from two specimens of the disease—the one a case under the care of Mr. Henry Morris, of the Middlesex Hospital, which I saw operated on in April, 1873 (Case 6 in the Appendix), and the other Case 2 in this paper. Both these examples had reached the point when ichthyosis becomes epithelioma, and both were for that reason submitted to operation. From the portions of the tongue removed in these two instances numerous sections were kindly made for me by Dr. Edward Sparks, and the appearances which they presented were marked by some differences of detail, especially with regard to the mode of invasion of the cancerous disease.

Where the section traversed only the ichthyotic coating the appearances were much the same in both cases. There was some increase in the thickness of the epithelial layer, though, perhaps, not so much as one might have anticipated. There was also some enlargement of the papillæ, and a great development of the rete mucosum. Around the bases of the papillæ and in the submucous and muscular tissues there was a very abundant nuclear cell-growth. There was also a notable increase in the number and size of the blood-vessels in all parts of the disease, in the non-cancerous as well as in the cancerous portions (Plate IV, fig. 1).

But where the indications of epithelial cancer were visible there were some differences between the two specimens. In the sections taken from Mr. Henry Morris's case the most striking feature was the remarkable development of the rete mucosum. It had increased enormously at the expense of the papillæ, reducing them, in many cases, to mere threads, and dipping down between them in the form of large club-shaped processes. Towards the termination of some of these processes the cells were assuming a circular arrangement, and forming the "laminated capsules," or nests of cells, that are so characteristic of epithelioma (Plate IV, fig. 2).

In the other specimen a few of these processes are visible, but they did not constitute the leading feature. For the most part the papillæ and their superficial structures, though enlarged and thickened, seemed to retain their original relation

to one another; but below them, in the submucous layer and among the muscular fibres, were numerous "laminated capsules" (Plate IV, fig. 3). Indeed, the number of these nests of cells which some sections presented was very remarkable. The club-shaped processes of the rete which were seen in this specimen were neither so numerous nor so largely developed as in Mr. Henry Morris's case. Perhaps these differences depend in some degree upon the age of the cancerous disease. The second case was operated on at a comparatively early date after the epithelioma had supervened. Or perhaps they may depend upon the precise point at which the cancerous disease commences, whether on the surface or in the deeper tissues.

I propose now to mention the particulars of a few cases, some of which have fallen under my own care, while others have been communicated to me by friends, or have been gleaned from various publications. In the Appendix I have noted, in a very abbreviated form, some other cases that have been obtained in like manner.

Case 1.—The earliest example that I have met with is a case recorded by Dr. Ullmann in the 'Bavarian Medical Intelligencer,' 1858, under the the title of "Formation of Callosities on the Tongue—Hypertrophy of the Epithelium of the Tongue—*Tylosis linguæ*." The author gives these names to a peculiar affection of the tongue, which he observed in an actor, aged 65 years. The disease had developed itself in twelve years without any assignable reason, unless the patient's habit of smoking very strong cigars could be considered a predisposing cause. There were on the surface of the tongue more or less intensely white plaques, between which the mucosa was of a dull colour, this tint blending towards the edges and tip with the normal appearance. In some places the patches were sharply defined. They could not be scraped off. They presented a hard consistence, and could only be cut away bit by bit with a knife. When this was done the papillæ looked red and increased in size, but under the thicker patches no papillæ

at all could be made out. On the front of the right half of the tongue these growths were most distinct, and surrounded an intensely red spot of the size of a kreuzer. On this spot there had previously been, according to the patient's statement, similar white growths, which had separated as a slough after a piece had been cut out. The border of this reddened spot was surrounded by a wall of from a quarter to half a line in height. After the removal of a few of the bits of white growth they always grew again. As the disease gave the patient no annoyance no active treatment was adopted.

Case 2.—Henry W—, æt. 38, first applied at Charing Cross Hospital in October, 1871. He had formerly been in the army, but was then a sale-room porter. Fourteen years before, he had contracted syphilis in India, and had subsequently gone through the whole series of secondary symptoms. His tongue had been ulcerated for eleven years; at first the ulcerations were confined to the sides, these he neglected and they spread over the dorsum. When I saw him the upper surface was covered with a white, silvery coating, in some places rising into hard elevations like corns. Some small spots on the under surface of the tip were ulcerated and tender. He said the disease was always worse when he had been drinking or exceeding in any way. He was an habitual smoker, though smoking made his tongue smart, particularly where it was ulcerated. The *corns*, he said, marked the places where the ulceration had been most severe, and where caustic had been most freely applied by himself and by the surgeons who had seen him.

This patient has continued under my observation for two and a half years. He has been treated with anti-syphilitic remedies as well as with other medicines, and mild caustics have been applied to his tongue. But the treatment has produced no marked effect. He was a man who lived freely, and who took but little care of himself. Sometimes his tongue has appeared rather cleaner, because he was living

regularly and his general health was better. At other times it has been worse, because he has been drinking to excess and disordering his digestion.

Things went on thus till last autumn. On November 14th, 1873, he came to me and said that about eight or ten weeks previously he had noticed a lump in the left half of his tongue, and that there was now so much shooting pain—there having been before only some local soreness where it was ulcerated—that he thought it must have assumed a fresh character.

On examining it I found a hard lump, about as large as a hazel-nut, in the left half of the tongue. It was situated about the middle of the free portion. On its dorsal aspect there was an ulceration about the size of a split pea. The surrounding tissues were soft and felt healthy. The general ichthyotic characters of the rest of the tongue remained unchanged. The patient had a haggard look, and complained of a great deal of sharp pain under the jaw and shooting towards the ear and the vertex. The glands at the upper part of the neck, behind the angle of the jaw, were slightly enlarged on both sides, but most distinctly on the left side. Under these circumstances I recommended him to have the portion of tongue including the painful lump removed at once. He readily consented, and on the 22nd of November I took away, with the galvanic écraseur, about a third of the free portion of the tongue. He made an excellent recovery, and left the hospital on the 9th of December, the raw surface granulating healthily, and the patient being entirely relieved from the sharp shooting pain that had before caused him so much distress. It was from the portion of tongue removed in this case that some of the microscopical sections already described were made.

But the relief thus obtained was of short duration. Before two months had elapsed it was evident that the other half of the tongue was invaded by the cancerous disease. An ulcer formed on a spot on the dorsum where there had been a particularly thick patch of ichthyosis; the anterior part of the organ became swollen and hardened, and an enlarged

gland beneath the chin became very apparent and very painful. As the back of the tongue remained soft and natural, and as there were no other enlarged glands to speak of, I recommended a second operation. But the patient hesitated and begged for delay, and a fortnight later, when he was willing to give his consent, the disease had made so much progress that I could no longer recommend it. [He died April 27th, 1874.]

CASE 3.—William H—, a butler, æt. 46, living at Boston, in Lincolnshire. Married. Has six children; one (the first) died in infancy, the rest are all healthy. His wife has miscarried two or three times. He is a healthy looking man, and says that he has generally been very well. He never had syphilis, and his tongue was never sore before the present affection came on.

In 1861 he had rheumatic fever. It lasted four months, and when he was recovering the *ichthyosis linguæ* first showed itself.

On May 24th, 1872, he was sent to me by Dr. Mercer Adam, rather as a matter of pathological interest than with a view to any active treatment. The following is the note which I made at the time:—He has now a symmetrical patch of thick fur on each half of his tongue. It is of snowy whiteness, and not unlike the rough side of white kid leather (Plate V, fig. 1). Sometimes, he says, it is thicker, sometimes thinner; sometimes it comes off in small pieces, sometimes it is so firmly attached that it hurts him to scrape it. The adjacent portions of the tongue are perfectly clean, so that the contrast between the healthy and the diseased parts is very striking. The white patches are raised about an eighth of an inch from the surface. His teeth are bad, and he has lost many of them. He has not had toothache; but they become loose and drop out, or he pulls them out. He attributes their loss to "the hundreds of bottles of medicine" that he has taken for the cure of his tongue.

The morbid patches were strikingly symmetrical. They

looked as if a white butterfly with outstretched wings had settled upon his tongue. Their snowy, almost woolly, appearance suggested the idea that a vegetable parasite might be present. But on careful microscopical examination no trace of such a thing could be found. Though the patches looked snowy, they were hard to the touch, indeed almost cartilaginous and firmly adherent.

The patient says that at first there was only a small patch on the centre of his tongue, about the size of a fourpenny piece. This gradually increased for six years, at the end of which time it had reached its present size and shape. From that date until now it has remained stationary. Both common sensation and the sense of taste are much impaired, though not altogether destroyed, on the affected surface.

In the spring of 1873 Mr. Pilcher, of Boston, under the impression that the disease must be syphilitic, gave the patient iodide of potassium with one twelfth of a grain of the red iodide of mercury in each dose. This treatment had a very marked effect, and on December 5th, 1873, Dr. Mercer Adam kindly wrote me the following note:—"The patient's general health has improved, and most of the thick white deposit has disappeared from the tongue. A largish patch still remains on the left side, the size of a shilling, but otherwise the organ is free from it. On the surface, where the deposit formerly existed, there are visible, here and there, little milky looking spots, almost of an aphthous character, which may be incipient patches of ichthyosis."

[This patient attended the meeting, and was examined by the Fellows present.]

Case 4.—William B—, æt. 63, formerly an auctioneer's foreman, but now an inmate of the Central Sick Asylum, at Highgate. To Dr. T. S. Dowse, the medical officer of that institution, I am indebted for opportunities of seeing the patient and making the following notes.

December 17th, 1873.—The patient is a hale old man, and says that he has generally had good health. When he was young he had gonorrhœa, but never syphilis, and on

questioning him closely no indications of a syphilitic taint can be discovered.

Two years ago he had "gatherings in his ears," accompanied by great pain and subsequent discharge, and his mind was slightly affected. It was then that his tongue first became diseased. At that time it was much more sore and painful than it is at present, for it was "quite raw," and there were some cracks upon it. These were occasionally touched with nitrate of silver. After that the white "skin" gradually came over it, and it has remained much the same ever since. It is always worse when his bowels are confined and when his digestion is at all deranged.

He still has obscure cerebral symptoms, numbness in the right arm, giddiness, pain in the head, as well as frequent discharges from his ears, especially from the left. When his ears are discharging he is free from pain in the head.

The whole of the dorsum of the tongue in its anterior part is covered with a thick white coating, wrinkled and corrugated. Towards the middle it slants off to the sides, so that the healthy mucosa comes forward, as it were, in a V shape (Plate V, fig. 2). The white appearance extends round the sides of the organ to the under surface, almost as far as the frænum, in milky white patches, and the same appearance is visible on the inside of both cheeks and of both lips. When the tongue was acutely sore his mouth was so tender that he could not take any salt, pepper, or mustard in his food. Now he can eat them all freely. Indeed, common sensation and taste are both blunted on the affected portion of his tongue. He has taken iodide of potassium and other medicines, but they have produced no alteration in the disease.

CASE 5.—The following case has been kindly communicated to me by Mr. Hancock.

Capt. D—, a spare man, æt. 53, had a small white warty excrescence, about the size of a split pea, upon the right side of the dorsum of his tongue. It had a hardened base rather

larger than itself. It gave the patient no pain; in fact, he was not aware of its existence till it was discovered by his ordinary medical man, when attending him for some trifling illness. The glands in the neighbourhood were not implicated. Mr. Hancock therefore removed the disease by a V-shaped incision. The patient did very well at the time, but seven years afterwards another growth of the same character appeared close to the cicatrix. The disease still being localised, Mr. Hancock again operated. On this occasion he removed a semilunar piece of the tongue, carrying the cut well beyond the hardened base. The patient lived five years after the second operation, and died of pneumonia. There was no appearance of any return of the tongue disease.

Case 6.—For the notes of this case I am indebted to Mr. James Adams, of the London Hospital.

Davison P—, æt. 39, first came under observation in April, 1872, with a large patch of ichthyosis on the right half of his tongue. He said that he had first noticed a small pimple under the right side of his tongue twenty-one months before. This gradually spread. It gave him no pain except during mastication and when smoking. He has never had syphilis.

The white patch appeared to consist of hypertrophied papillæ, covered and surrounded by very thick epithelium. The subjacent muscular tissue was perfectly soft and flaccid, while the thick covering felt like leather that had been soaked in water and then dried.

Iodide of potassium was ordered for some time, and subsequently arsenic, but the tongue manifested no improvement. At the end of twelve months the patch had increased in size and thickness, and looked as if it was about to ulcerate.

Unfortunately this patient has not been seen since.

Case 7.—Dr. J. Moore Neligan has related a most interesting case in the 'Dublin Quarterly Journal of

Medical Science' for August, 1862. The following is an abbreviated account of it:—

H. E—, æt. 46, appeared before Dr. Neligan, on the 17th of April, 1857, to be examined for life assurance. In his paper he stated that he had never had any illness since childhood, and that he never had occasion to consult a medical man. His family history was good, and his own health seemed to be excellent. But his tongue was singularly affected. "The natural membrane covering it and the inside of the cheeks being changed into a thick white skin, like a kid glove." He said that it had been so for the last thirty years, that his taste was as perfect as that of any other person, and that he had no soreness or uncomfortable feeling in it.

The tongue was perfectly clean—that is to say, there was no fur upon it, nothing that could be removed by scraping or washing. It was of a dead white colour, resembling, perhaps, rather the tongue of a boiled calf's head than a kid-skin glove, the lustre of which it wanted. It was uneven on the surface, but not wrinkled or fissured, nor did it present the papillated character of the organ in its normal state. There was more a general unevenness. The same condition existed in the mucous membrane lining the cheeks and the gums in contact with them, but the covering was evidently less thick. The roof of the mouth, the palate, the throat, the tonsils and the uvula, were quite natural in appearance. On closely questioning the patient he stated that he had noticed this change when he was about eighteen or nineteen years of age, and that then it was just as complete as when he was first seen by Dr. Neligan. He thought when he first discovered it that it must have been caused by smoking to excess, and by a habit he had of always smoking the tobacco in the shortest possible pipe, so as to get the smoke into his mouth as hot as he was able to bear it.

This gentleman was seen by Dr. Neligan from time to time up to the 3rd of June, 1861, and the most careful examination failed to detect the slightest alteration in the state of the tongue.

About the end of September, 1861, the patient accidentally bit his tongue. The result was that a small tubercle, about the size of a pea, formed on the edge, beneath the mucous membrane, its situation being on a level with the molar teeth. This gradually assumed a cancerous character. The glands in the neck became affected, and the patient died in a few months.

In this case we notice particularly that the disease came on at a comparatively early age, and lasted over thirty years, when a very slight cause determined the commencement of epithelial cancer.

As another example of the disease commencing early and continuing through many years, I may mention a case which I was asked to see in March, 1873, by Mr. Francis Fuller, of the St. Marylebone Infirmary.

Case 8.—John H—, æt. 68, was an in-door servant till about five years ago, when he caught a violent cold. This was followed by rheumatism and chronic bronchitis, on account of which he was admitted into the Infirmary.

He says that he has always been troubled with indigestion. Forty-five years ago he used to have a foul tongue, which he was in the habit of scraping with a tongue-scraper. He cannot tell when the leathery coating which now overspreads his tongue first appeared. He believes it has existed ever since he was twenty-one, but during the last fifteen or sixteen years it has been getting worse. When he was young he had gonorrhœa, but he never had any symptoms of syphilis.

When I first saw him (March 28th, 1873) the dorsum of the tongue was entirely covered by a thick, white, persistent fur. It was wrinkled and chipped, so that it had the appearance of being divided into scales. Two months before, in consequence of strong applications which were made to the tongue at a special hospital, a patch of the fur came off and left a bare spot, about the size of a sixpence, half an inch from the tip. This spot was red and raw, and so sensitive and tender that he could not take any mustard, pepper, or

vinegar with his food. Near the tip the white appearance turned round the edges of the tongue towards the under surface. At this date I noticed no induration in or around the raw spot.

On May 26th, 1873, the following note was made:—The raw spot has grown up, forming a tumour about the size of a bean, and in front of this is a little ulcerated pit. The anterior part of the tongue seems altogether swelled, and there is a constant flow of saliva from the mouth. The ichthyotic coating is very thick towards the back, behind the ulcerated spot, and of a yellowish colour. The lump and the ulcerated spot are both hard, and the induration extends to a considerable area around. The submaxillary glands on both sides are somewhat enlarged. The patient complains of pain under the jaws and towards the ears and the vertex.

As it was evident that the disease had now become epitheliomatous, I proposed to remove the whole of the anterior part of the tongue, but the patient was unwilling to submit to any operation.

On July 3rd he came to me again. The hard lump was then sloughing in its centre, and it was very sore when touched. He now complained of pain down the neck and up the side of the head. The submaxillary glands were enlarged, but not the lymph-glands. The operation was again urged upon him, but in vain.

Soon after this the patient took his discharge from the Infirmary, and went for advice to several hospitals—at length being admitted into the Middlesex on August 5th, under the care of Mr. Hulke. By this time it was judged too late to perform any operation. [He died March 14th, 1874.]

Case 9.—In a case related by Dr. Church, in the first volume of the 'St. Bartholomew's Hospital Reports' (1865), an ichthyotic condition of the tongue was associated with patches of the same nature in other parts of the body. The patient was a delicate girl, aged 15; and the disease, which was congenital, was vaguely attributed by the mother to a fright she had had during her pregnancy.

The following is an abbreviated account of the case:—The affection of the skin was confined to the left half of the body, scarcely crossing the middle line at any point. The disease presented two distinct characters, being in some places papilliform, in others squamous. The skin on the left side of the trunk was everywhere of a darker hue than that on the right, a well-defined line being visible down the centre of the thorax and abdomen.

A large patch covered the left side of the forehead and nose, the affected skin being slightly papillary in character. A patch similar in appearance spread over the greater part of the cheek and chin on the same side, and extended from the face to the tip of the ear.

The whole of the left side of the neck was occupied by the disease, which here assumed a warty character, some of the papilliform outgrowths being pedunculated. The diseased portions of skin stopped abruptly, both before and behind, at the middle line of the neck.

The scapular region was almost entirely occupied by a large patch of a steel-grey colour, very slightly elevated above the surrounding skin, and consisting of small polygonal scales. The skin, so altered, felt quite smooth and soft to the touch, could be easily pinched up between the fingers, and nowhere exhibited traces of the cracks and fissures usually described as present in ichthyosis. The greater part of the left half of the chest was affected in a similar manner, the areola of the nipple being the seat of long conical papillæ of a browner colour than the surrounding squamous portions. At the border of the axilla, and in the axilla itself, the papillæ were larger and darker than in any other part of the body. Smaller patches, similar in every respect to that on the chest, existed on the subscapular and lumbar regions, a very small one at the commencement of the anal fissure, and another larger one on the buttock. Nowhere, not even in the neck and axilla, where the papilliform masses were largest, was the integument stiff or rigid, while the non-elevated squamous portions were quite remarkable for their softness.

A similar change had taken place in the mucous membrane of the mouth, whilst that of the eyes, nose, and vagina was unaffected. The whole of the mucous membrane covering the inside of the left cheek, and the left half of the soft palate and tongue was the seat of papilliform outgrowths, which closely resembled those on the neck and in the axilla, but contained no pigment, and were of a dull yellowish-white colour.

This patient died suddenly, and at the *post-mortem* examination it was found that the heart was greatly hypertrophied, that the foramen ovale was slightly open, and that the aorta immediately below the origin of the left subclavian artery became suddenly narrowed.

The fact that both the cutaneous and the mucous surfaces were affected, and that the morbid condition was limited to one half of the body, are the points which are the most remarkable in this case as bearing upon our present subject. As this case was of congenital origin, it ought clearly to be placed in a separate category from the examples of *ichthyosis linguæ* that have been already described.

In studying these detailed cases, as well as in looking at the Appendix, there are several points which strike us as worthy of special notice.

1st. The great preponderance of cases in which *ichthyosis linguæ* affects men. If we exclude Dr. Church's case, which seems to belong to a separate category, there is only one female patient in a list of sixteen cases. It would appear, therefore, that the disease which is properly the subject of this paper—a morbid condition which supervenes upon a previously healthy mucous membrane, and which has a strong tendency to become epitheliomatous—is almost entirely confined to men.

2nd. *Ichthyosis linguæ* is, in its commencement, a disease of early manhood and middle age. It never occurs before puberty. I have, however, seen a case which formed an apparent exception to this rule, and which it may be worth while to mention.

A little girl, æt. 3, had on her tongue several sharply defined, snow-white, elevated patches, which presented all the appearances of ichthyosis. But after a time they lost their sharp edges and white surface, and were overspread by a foul yellowish secretion. It was evident then that they were merely mucous tubercles; and under appropriate treatment they got well.

3rd. We notice that *ichthyosis linguæ* is occasionally associated with syphilis. This naturally leads us to ask, Is it always syphilitic? Is it merely one of the many manifestations of that Hydra-headed monster? Is it a form of disease claiming the same origin as the mucous tubercle, and more analogous to it than to any of the other diseases that have been named in the earlier part of this paper? To this I reply that, though it is sometimes due to superficial syphilitic ulceration (as in Case 2), this is only one among various causes that are capable of producing it. In many instances no syphilitic evidence whatever can be obtained. In none, as far as I know, has any complete or permanent amendment been brought about by anti-syphilitic remedies, no matter how early they were employed, or how slight was the case. Again, the disease has a strong tendency to become epitheliomatous, and will assuredly, if the patient live long enough, develop into epithelial cancer. But such is not the history of ordinary syphilitic sores or growths. For these reasons it appears to me that we are not warranted in saying that the disease is one of syphilitic origin. On the contrary, it is clearly distinguished from the manifestations of syphilis; though, as we have seen, a venereal ulceration may be its starting point. In persons of a peculiar idiosyncrasy it appears as if any oft-repeated or long-continued irritation of the lingual branches of the fifth pair of nerves were capable of exciting the disease.

With regard to treatment we observe that the most active medicines have been given—mercury, arsenic, iodide of potassium, &c.—but they have failed to effect a cure. The most powerful caustics have been employed, and yet they have not removed the disease. I have a strong opinion that

the best we can do for these patients is to study their general health, and that if any local measures are used they should be of an unirritating kind. When a case presents itself in an early stage with the ichthyotic patch no larger (let us say) than a fourpenny piece, my advice is to excise it freely and at once. The result which Mr. Hancock obtained by following this course is very encouraging (Case 5). Again, when the disease is evidently becoming epitheliomatous, no time should be lost in undertaking an operation; for though the results of such operations are not satisfactory as regards an ultimate cure, there can be no doubt that they both prolong the patient's life and render his existence more tolerable. But between the early and the late stage of the disease I hold that the less the tongue is meddled with the better. I am opposed to all caustic applications as well as to all cuttings, parings, and scrapings. If there are any teeth which seem likely to injure the tongue they ought to be extracted. At the same time the patient should be careful about his diet, avoiding everything that makes his tongue smart; and above all he should pay proper attention to his digestion. It is in this direction that the surgeon can be of most use during the middle period of the disease, by giving such advice as will guard the tongue against all sources of irritation, and will regulate and improve the general health. I have often been struck by the way in which the ichthyotic coating becomes altered for the better under the careful regulation of the stomach and bowels, though it never quite disappears. In cases such as these our duty is to reduce the evil to a minimum if we cannot altogether remove it.

APPENDIX.

Case 1.—Mr Hulke reports a case in the 'Clinical Society's Transactions,' vol. ii, p. 1. The patient was a man, æt. 43. He was first seen in February, 1861. There

had been ichthyotic patches on his tongue for twelve or fourteen years. He never had syphilis. A small patch was cut off, and free bleeding followed. In April, 1864, a larger piece was removed with the écraseur; and in May of the same year another patch was cut off with the scalpel. In December, 1867, he had a ragged ulcer, with raised margin and thickened base, near the tip of the tongue. The tumour was removed with the écraseur. In the following June he was last seen, and was then evidently near his end.

Case 2.—This case as well as No. 3 have been kindly communicated to the writer by Mr. Hulke.

A printer, æt. 42, was admitted into the Middlesex Hospital on August 12th, 1870. Scattered over the upper surface of his tongue were several leathery plaques of thickened epithelium of very old standing; and in the left side was a puckered excavated ulcer, the edges and base of which were indurated. Below and behind the body and the angle of the lower jaw, on the left side, were two large nodular masses, red, prominent, and fluctuating, one of them being superficially ulcerated. The tongue was fixed by extension of the infiltration from around the ulcer into the floor of the mouth. He said that the ulcer began about nine months before as a small hard crack, which he attributed to having bitten his tongue. Towards the end of August the cluster of enlarged glands under the body of the jaw broke, and soon became a deep, hollow, sloughy ulcer, with undermined and everted edges. From this time he rapidly sank, and died on October 10th.

Case. 3.—A bronzed-face, hale-looking farm-labourer, æt. 50, was admitted into the Middlesex Hospital on August 19th, 1871, with a deep, narrow ulcer in the left half of his tongue, grooving it from the tip nearly to the base. On both sides of the tongue were several opaque plaques of a whitish or buff colour, manifestly restricted to the surface, unsurrounded by any hardness, the tissues immediately limiting them appearing perfectly healthy. They had ex-

isted, the patient said, very many, he did not know how many, years. He first noticed the ulcer in the preceding May.

Case 4.—Sir James Paget has reported the following case in the 'Clinical Society's Transactions,' vol. iii, p. 88:—The patient was a lady, æt. 42, and was first seen in August, 1869, on account of patches of ichthyosis on the right side of her tongue. They had been increasing for twelve months. They occupied only the papillary structures, and had no indication of cancer in or near them. In December, 1869, thickening and hardening were first observed in and beneath the ichthyotic patches, and in another month the whole side of the tongue was occupied by well-marked ulcerated epithelial cancer. In this patient there was an hereditary tendency to cancer.

Case 5.—In St. Bartholomew's Hospital Museum ('Cat.,' vol. i, Ap. 4) there is a tongue in which the anterior three fourths of the dorsum are occupied by a circular ulcer, with a broken and shreddy surface. The margin of the ulcer is nearly surrounded by a hard layer of opaque white epithelium, which is in parts a line in thickness. Around this layer the tongue appears healthy. The patient was a man, aged 68; the disease commenced eight years before death in a small, hard, white lump in the middle of the dorsum of the tongue, on a spot upon which the end of a tobacco-pipe had often rested. The patient was for several years in the habit of paring this lump with a razor twice a week. It enlarged and extended all over that part of the tongue now occupied by the ulcer, but it gave him no inconvenience except from its hardness. About four months before death ulceration commenced, and extended over nearly all that part of the tongue that had been covered by thickened epithelium. The organ at the same time became very large, completely preventing deglutition, and the patient died exhausted.

For notes of the three following cases I am indebted to Mr. Henry Morris.

Case 6.—Edwin T—, æt. 50, applied at the Middlesex Hospital on December 27th, 1872. He had had syphilis seventeen years before, but his tongue had never been sore previous to the commencement of the present disease. He has been a great smoker.

For the last nine or ten years the greater part of the dorsum of his tongue has been overspread by a white, leathery coating, arranged in a somewhat symmetrical manner on each lateral half of the organ. Upon the left side of the tip was a raised irregular mass, about the size of an almond, quite hard, and fissured on its surface. There was no enlargement of the lymph-glands. For four or five weeks anti-syphilitic remedies were prescribed, but without effect. Subsequently a small, hard nodule appeared on the right side of the tip, and an enlarged gland was noticed at the left angle of the jaw. There was also more pain than before.

On April 3rd, 1873, the anterior half of the tongue was removed with the galvanic écraseur, and the patient made a good recovery.

Case 7.—William C—, æt. 49, was admitted into the Middlesex Hospital, under the care of Mr. De Morgan, in April, 1873. He never had syphilis. For the last ten or eleven years there had been a white, horny patch upon the left side of the dorsum of the tongue. Nine months before admission this spot began to ulcerate, and was now occupied by a superficial ulcer. There were no enlarged glands in the neck. On the lining membrane of the cheeks there were some white patches.

On April 23rd Mr. De Morgan sliced off the superficial ulcer with scissors, and the patient left the hospital on May 27th.

In August he was readmitted, with a hard nodule just behind the cicatrix, and pain extending towards the ear.

On September 3rd, 1873, the right lingual artery was tied by Mr. Morris, and then the diseased structures were

Fig. 1.

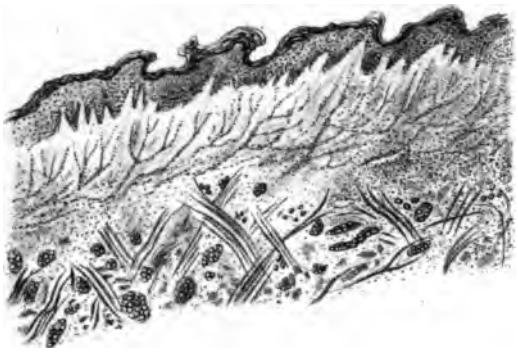


Fig. 2.

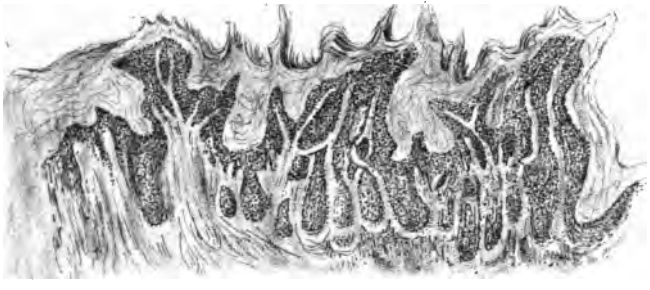
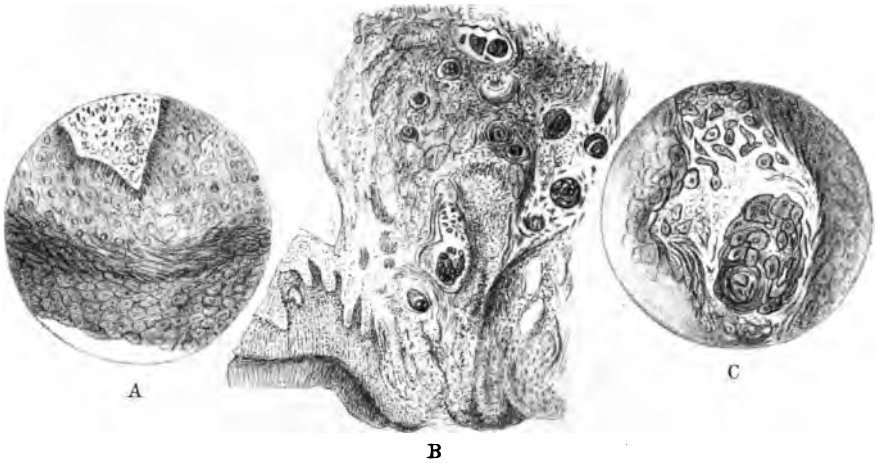


Fig. 3.



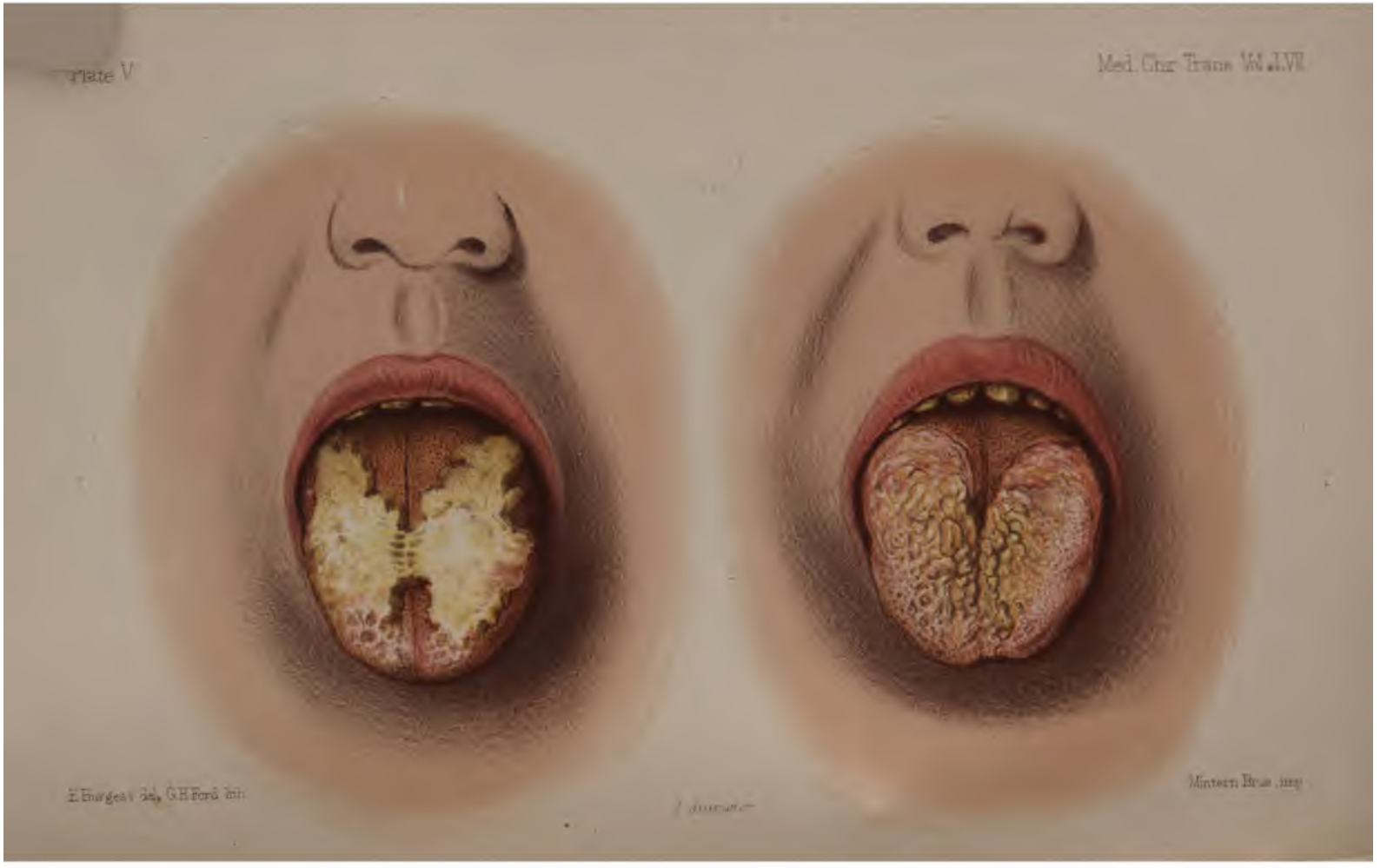


Plate V
Med. Chir. Trans. Vol. LVII
E. Burgess del. G.H. Ford lith.
Mintern Bros. imp.

freely removed with the knife. The patient was discharged, well, on September 23rd.

Case 8.—Samuel B—, æt. 56, applied at the Middlesex Hospital on May 1st, 1873. He never had syphilis. For at least ten years he has had tough white patches on his tongue. Eight months ago a small elevated ulcer commenced on the right side, near the tip. When first seen, there was a ragged ulcer extending from the tip nearly to the base of the left side of the tongue. On the right side there was an ichthyotic patch, which extended towards the under surface. There was one enlarged gland beneath the angle of the left jaw. The patient refused to undergo any operation.

These three cases are reported in full in the 'British Medical Journal' for February 21st, 1874.

DESCRIPTION OF PLATES IV AND V.

PLATE IV.

FIG. 1.—Ichthyosis linguæ. × 40. Showing the thickening of the epithelial layer, the development of the rete mucosum, and the great increase in the number and size of the blood-vessels (see page 159).

FIG. 2.—Ichthyosis linguæ, with epithelioma. × 40. (H. Morris's case.) Showing the club-shaped processes of the rete, which has developed at the expense of the papillæ (see page 159).

FIG. 3 (see page 160).—*a*. Tip of left-hand papilla. × 200. Showing the great thickness of the rete and of the epithelial layer.

b. Ichthyosis, with epithelioma. × 40. Showing the nested cells in the submucous tissues, but no processes of the rete.

c. The central nest of cells. × 200.

PLATE V.

Icthyosis Linguæ.

FIG. 1.—Case 3. William H— (see page 163).

FIG. 2.—Case 4. William B— (see page 165).

ON THE

AMOUNT OF CARBONIC ACID

FOUND BY EXPERIMENT

IN THE AIR ON BOARD WOODEN FRIGATES.

BY

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SURGEON OF H.M.'S SHIP "DORIS."

COMMUNICATED BY
DR. SYMES THOMPSON.

(Received February 10th—Read March 10th, 1874.)

THE Doris is one of six wooden frigates forming the Detached Squadron, and at present cruising on the West Indian Station. In a ship of this class, with numerous well-arranged windsails, large hatchways, ports and scuttles—provided all these can be kept open, and the windsails are properly trimmed, the ventilation upon the whole is pretty good; at any rate, it is much better than has been generally supposed.

Numerous carefully conducted experiments I have found most decidedly to point in this direction.

This free and somewhat efficient ventilation of our ships-

of-war is the normal state of affairs just where and when it is needed, viz., in harbour in the tropics. Here, as a rule, ships are a good deal at anchor, and there is really nothing to prevent those in authority from taking full advantage of the somewhat ample means of ventilation at their disposal. But when the rainy season sets in, so many parts of the ship are occasionally obliged to be shut up which during fine weather are always kept open, that the ventilation is apt to become very defective indeed; and the air becomes loaded with all the usual impurities which are always found in overcrowded and confined places. Yet under these unfavorable conditions I have not found that the amount of carbonic acid present in the air on board ship is in excess of that of overcrowded ill-ventilated rooms on shore.

In cold, and temperate climates, too, there is too apt to be a good deal of shutting up of what may be called the natural ventilators of a ship. Nor are windsails made use of quite so frequently as they ought to be, on account of the desire which so generally exists to shut out the cold fresh air. In stormy weather, in the English Channel, on account of almost everything in the shape of an opening being closed, the air on board ship of course becomes greatly vitiated, more especially in the lower parts of the ship. But this is a condition of affairs which is happily somewhat exceptional; if it were otherwise our sailors would suffer terribly.

It must always be borne in mind that the boys for the Royal Navy, who become our seamen as they grow up, are picked from a large number; only the healthiest and most robust being chosen as fit for the service; large numbers being rejected who do not quite come up to the required standard either in physique or measurement, but who nevertheless probably become very useful members of society in other walks of life. We therefore have a right to expect a high standard of health and physique amongst our seamen of the Royal Navy, seeing how very carefully they have been selected. Therefore, when drawing comparisons between our sailors and people on shore of a similar social class, these facts should always be borne in mind.

For instance, I believe that chest complaints are not more common amongst our seamen afloat than they are amongst people of a similar class of life on shore; whereas one might naturally expect to find a far larger percentage of these affections on board ship, on account of our sailors having to breathe a somewhat vitiated air between decks. But, as I have stated before, as these are all picked men, no useful comparison can be drawn between them and persons living on shore. Moreover, most of the chest complaints of the Navy happen, not amongst our fighting seamen proper, but amongst the stewards and mechanics, who pass their time chiefly down below, and who are not very carefully selected on their first entry, on account of the very different nature of their duties from those of seamen.

Again, the sailors of our Royal Navy and those of the merchant service, with regard to sickness and disease, do not admit of any useful comparison, on account of the very different circumstances which obtain in the two services. In the merchant marine there is not much attention paid to medical examinations of men prior to their entry for a voyage, nor is cleanliness so much attended to as it is in the Royal Navy. Also, in the merchant service, if a man breaks down in health, he can always obtain employment again when he is sufficiently recovered. It is not so in the Royal Navy, for if a man is once invalided out of the service, he can never be employed afloat again.

Ventilation is another matter that is not nearly so well looked after in our merchant ships as it is in ships-of-war. So that in the one service we have men carefully selected, and well cared for in the important matters of food, clothing, cleanliness, and ventilation; whereas, in the merchant service, these things are not nearly so well looked after. In the Royal Navy there is always at least one medical officer to advise the executive about all sanitary matters.

For these reasons the percentage of sickness amongst our merchant seamen must necessarily be in excess of that which exists amongt the men of the Royal Navy.

As seamen pass so large a portion of their time on the upper deck, they get rid, in a great measure, of the ill effects produced by breathing the impure air below.

With regard to the cubic space required on board men-of-war, to give a fair chance of health to our sailors, 300 cubic feet has been fixed upon as a minimum; but on account of the limited space at our disposal, even this small amount of breathing room can rarely be allowed; whereas on shore 2000 feet is considered necessary, and 800 feet for soldiers in barracks.

In the Doris—a wooden frigate, and one of six such ships at present (May 18th, 1873) forming the Detached Squadron, and cruising in the West Indies—121 cubic feet represents the sleeping accommodation for each man berthed on the lower deck, and it is here that the larger portion of the crew sleep.

The Doris carries 24 guns, is 2483 tons, and has 800 horse-power. She was commissioned at Devonport in October, 1872, for service with the Detached Squadron. Her complement is 490 officers and men, but with supernumeraries we have generally numbered about 550. She is a good sea-boat, but rolls excessively, probably on account of her massive machinery and heavy guns. She has great beam, very good height between decks; her ports are unusually large, and she has very large hatchways. The men sleep on the main and lower decks.

The main deck, after all the necessary deductions, has an available cubic space of 51,586·1459 cubic feet, and as there are 176 men and boys sleeping on this deck at night, each individual has therefore a space of 287·102 cubic feet.

The lower deck, after all the necessary deductions, has an available space of 42,960·02580 cubic feet, and as the number of the men berthed on this deck at night is 353, each man has therefore 121·7 cubic feet of space. These are the numbers at the present time, but they vary to a small extent occasionally.

Now, as all the ship's company mess on the lower deck, I

have calculated that during meal-times each man and boy has only 80·211 cubic feet of space.

In the sick-bay (situated on the main deck forward), after all necessary deductions for furniture, &c., there is an available space of 1270 cubic feet; it contains sleeping-room for eight people, so that each patient would thus have a space of 158·75 cubic feet.

In the gun-room, where twenty junior offcers live, there is an available space, after deductions, of 1000 cubic feet, so that each of these officers has but 50 cubic feet of space during the day. At night these officers sleep on the after-part of the lower deck, a place called the steerage, and then enjoy a space of 121·7 cubic feet.

The engineer officers' mess-place has an available space of 432 cubic feet, and as the number of these officers is four, each has during the day a space of 108 cubic feet. They also sleep in the steerage, and enjoy a space of 121·7 cubic feet at night. The warrant officers have much the same cubic space each as the engineers have.

The cells have a space of 175·168 cubic feet each.

Each of the senior officer's cabins, after deductions, has a space of 290 cubic feet.

So that, so far as sleeping room goes, each man and officer aboard has a breathing space of 121·7 to 290 cubic feet. This undoubtedly is a very small allowance, and far less than what is considered necessary for soldiers in their barracks on shore, in order to give them a fair chance of maintaining their health. Still, on board of a man-of-war the majority of the men are very frequently on the upper deck inhaling a very pure air, and a little of this occasionally taken into the system does away with a good deal of the previous mischief caused by overcrowding in a vitiated atmosphere below.

After numerous experiments made while cruising in the West Indies, and also during a passage from Madeira to Barbadoes, repeated over and over again in all parts of the ship, carbonic acid was the great impurity found in the air between decks and in the lower parts of the ship; the amount varied

at different times and places from 1·03 to 3·21 volumes per 1000, as shown in the accompanying tabulated statement. It was always most abundant on the lower deck and the deeper parts of the ship—in fact, in the worst ventilated parts, and where the men are most crowded at night.

On the upper deck even the amount of CO_2 was above the normal, and this evidently was caused by the heated and vitiated air coming up from below; instead of being only 4 volumes in 10,000, it rose as high as 8 and 9 volumes in the 10,000.

In air expired direct from the lungs into a receiver I found from 54 to 58 volumes of CO_2 in 1000. This was done sometimes to test our apparatus, &c.

Ozone was never present in large quantity, except on the upper deck; there was seldom the smallest trace of it on the lower deck, or in the parts of the ship below that. On the main deck there was a small amount present when the ports were open. I find the amount of carbonic acid in the air on board ships-of-war to be very nearly the same as exists in the air of the most crowded and worst ventilated places on shore, such as workshops, schoolrooms, &c. I have never found the enormous amounts of 18 to 33 volumes per 1000, as recently stated, to exist in the air on board frigates. Such statements seem to me to contain some element of error, as expired air alone contains but 50 to 54 volumes of carbonic acid in 1000.

Recent examinations of the air of public schools in New York show the existence of 0·97 to 3·57 volumes of carbonic acid in 1000. These numbers correspond very closely with those resulting from my examinations of the air on board the Doris, as will be seen by a glance at the accompanying table.

The large amount of carbonic acid existing always in the air on board ship must be injurious to the health of our sailors. "For," as Pettenkofer observes, "more oxygen is required and taken into the system during the night than in the day. But here we have a deficiency of oxygen, and hardly a trace of ozone present; and this state of things is

accompanied by the presence of twice or thrice to nearly ten times the normal amount of carbonic acid.

It has been proposed to work an electrical machine occasionally on board ship, to make up the deficiency of ozone in the deeper parts, but it seems to me this might do so merely at the expense of the already greatly reduced supply of oxygen.

All the above experiments taken together, exclusive of those made on the upper deck in the fresh air, give an average of 1·76 volumes of carbonic acid in 1000 volumes of air on board the Doris.[1]

Experiments showing the amount of carbonic acid found in the air on board H.M.'s ship "Doris."

Date.	No. of volumes of CO_2 per 1000.	Part of the ship examined, with the attendant circumstances.
1873.		
Feb. 15	2·24	Officers' cabin, wardroom, scuttle in, 8 a.m.
„ 17	1·03	Lower deck, midnight.
„ 19	3·21	Wardroom, 11 p.m., calm, ship rolling and scuttles in.
Mar. 2	2·4	Cell in fore-cockpit, with a prisoner in, 6 a.m.
„ 14	1·3	Sick-bay, ports closed, daytime.
„ 15	1·2	Weather side of main deck in screened berth, full of enteric fever cases, ports closed on this side.
„ 17	·9	On the upper deck, midnight.
„ 18	·8	On the upper deck, midnight.
April 20	1·6	Lower deck, in harbour, scuttles out, midnight.
„ 21	1·6	Do., upper part, in harbour, scuttles out, midnight.
„ „	1·35	Do., lower part, in harbour, scuttles out, midnight.
„ 22	1·7	Lower deck, in harbour, scuttles out, midnight.
„ 23	2·05	Wardroom, in harbour, midnight.
„ 30	1·5	Lower deck, in harbour, scuttles out, midnight.
„ „	1·4	After-cockpit, windsail down, midnight.
May 3	1·7	Fore-cockpit, no windsail, midnight.
„ „	1·1	Lower deck, foreward, midnight.
„ „	2·6	Officers' cabin, in wardroom, midnight.
„ 9	1·3	Wardroom, scuttles out in cabins, daytime.
„ „	1·2	Do., scuttles out in cabins, fresh breeze, midnight.
„ 11	1·3	Do., scuttles out in cabins, fresh breeze, midnight.

[1] Air containing 1·0 vol. of carbonic acid to 1000 is close and fusty; above this it becomes disagreeable. Air containing less than 0·6 per 1000 volumes is not perceptibly impure to the senses. (*Vide* Parkes, 'Hygiene,' 3rd edition, pp. 119, 120.)

A CASE

OF

NECROSIS OF THE JAW AND OTHER BONES

FROM THE FUMES OF PHOSPHORUS.

BY

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(Received March 1st—Read March 10th, 1874.)

Upon the table are the jaws and some adjacent bones of a lad, æt. 18, who had been exposed for a long time to the fumes of phosphorus.

He had been employed in the match trade for several years; from 1868 to 1871 in carrying fresh matches, and from the latter date to the end of 1872 in preparing phosphorus paste.

He was small for his age, barely five feet high, weighing a little over five stone, and looking much younger than he was. The mischief appears to have commenced about the middle of December, 1872, when the foreman noticed that the lad seemed to be ill, and shortly afterwards he could no longer work on account of pain in the jaw, which he referred to toothache. In January a molar was extracted, but without

relief, and the pain continued up to the date of his admission into the hospital, on the 29th March, 1873. At that time he was suffering greatly. The whole face, but especially the region over the lower jaw on the right side, was very painful, tender, much swollen, and the tense skin was pale and shining. The gums also of both upper and lower jaws were unduly vascular and very tumid. These symptoms increased until at lengh there was suppuration, and then, on April 14th, the lower jaw was found bare and evidently in a state of necrosis. After this he appeared for a while to be relieved, but soon the acute pain, swelling, and other symptoms were renewed in the region of the upper jaws; first on the right side, and soon after on the left. There was much suppuration here also, and at length it was evident that these bones were dying or dead. From time to time several loose teeth were removed. During the whole of this period he was very ill; his pulse varied from 100 to 120, occasionally even to 150 or 160, and his temperature during the more active mischief from 102° to 103°. Full doses of morphia were necessary to secure some sleep, and he became much emaciated. Occasionally, too, he suffered from obstinate diarrhœa. The suppuration was during the greater part of the time profuse, and the fœtor intolerable. This, even with the utmost care, could be only partially controlled by antiseptics.

On the 1st of August, six months before death, it was decided to attempt the removal of the lower jaw. The bone was divided in front, and each half was drawn out without difficulty and with scarcely any hæmorrhage. At this time there was not sufficient firmness in any part of the region to indicate the formation of new bone; but in the course of a week or two afterwards there was distinct evidence of new bone on either side about the angle, which gradually extended.

The patient rallied remarkably after the operation. The discharge was much reduced, and for a while he gained flesh and strength. There was no apparent diminution of sensibility in the skin of the chin. But in September severe pain returned, with abundant discharge and diarrhœa. There

were three or four sinuses in the cheeks between the orbits and mouth, and the whole of the hard palate was exposed. From this period until his death his condition varied at intervals. For some days he would be comparatively free from pain, sitting up and even cheerful. Then he would be entirely prostrated by a fresh accession of suffering and suppuration, and thus he was gradually worn out, and sank exhausted on the 22nd of January, 1874.

The following bones were denuded of periosteum and dead:—

On the right side.—The whole of upper maxilla with the central incisor; the malar; the external and internal angular processes of frontal, and the part in contact with the lachrymal and nasal bones; the lachrymal and nasal bones except at base; all the internal pterygoid plate and the front part of the external pterygoid plate of sphenoid, and a narrow strip of the orbital plate of the greater wing adjacent to the malar; all the palate bone; the inferior spongy bone, and the back part of the middle one; the os planum of ethmoid, which, however, was whiter than the other bones.

On the left side.—The whole of upper maxilla, with all the teeth; the orbital surface, especially at the back part, being less stained; all that part of malar adjacent to upper maxilla; the portion of palate bone attached to the specimen (see woodcut), with the exception of the orbital process; the inferior spongy bone; the lachrymal, which was less stained above than below; the nasal except at base; the os planum of ethmoid, which was whiter than the other bones; the vomer, except a strip in middle third of right side, which was still covered; below it was quite separated from the crest of maxilla and palate bones; the perpendicular plate of ethmoid behind.

In some parts the surface of the bones has a worm-eaten appearance, and in others is coated with a rough deposit. —The whole of the lower maxilla, which was removed during life, including even both condyles, with the two molar teeth on the left side. The enamel has been removed from the grinding surface of one of these, which appears

as if porous. Some portions of the front surface of the jaw especially of the right ramus, are coated with the pumice-stone deposit.

The new lower jaw which has been formed is shown (see figs. 1 and 2).

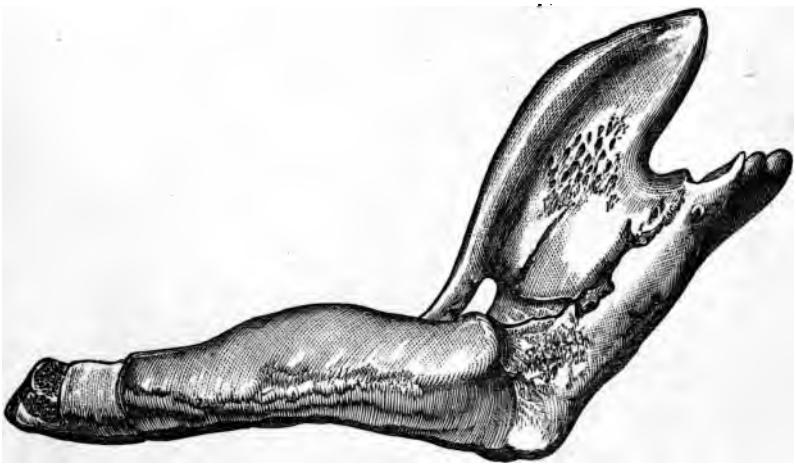
FIG. 1.

This is perhaps one of the most perfect specimens of the kind ever seen. In size, shape, and development, it is very remarkable. The bone is solid and dense, and in two pieces

only. The greater portion constitutes the whole of the bone, with the exception of the right ramus. This was united to the body by fibrous tissue, and separated during maceration. In size and form, and especially in the absence of alveolar portion, the jaw very nearly resembles the edentulous maxilla of a very old person.

Fragments from the surface of the left angle removed for examination differed in structure from adult bone chiefly

FIG. 2.



in the want of well-marked canaliculi and of well-defined laminæ. No doubt in deeper portions the bone is yet more perfect in structure.

The remarkable features of this case are: the extent of the necrosis, and the degree to which the lower jaw has been reproduced.

The specimens belong to the Museum of St. Bartholomew's Hospital.

ON

AN IMPROVED METHOD

OF

ABSCISSION OF THE ANTERIOR PORTION OF THE EYEBALL.

BY

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(Received March 18th—Read March 24th, 1874.)

In certain cases of staphyloma of the cornea, and in other forms of spoiled eye, it is sometimes desirable to remove the anterior portion of the eyeball. In every instance in which there is any likelihood, or even any possibility, of the presence of disease of the deeper parts of the eye of a kind which might produce sympathetic ophthalmia of its fellow, it becomes necessary to enucleate the diseased organ as a whole, leaving a stump to be formed by its muscles only. But where this danger does not exist it is better to remain satisfied with the removal of the front portion. The posterior half or two thirds of the eyeball will form the nucleus of a comparatively large and movable stump, on which an artificial eye can be placed with greater advantage than on a stump composed of muscles alone.

In former times it was customary simply to cut off a corneal staphyloma, and to leave the rest of the eyeball to shrink and cicatrize in its own time. Mr. Critchett greatly improved on this method by the operation commonly called after his name. He transfixed the eyeball by three or four semicircular, threaded, needles, excised the portion in front of them, and then drew the needles through and tied their threads in such a manner as to unite the gaping wound in a horizontal line. The sutures passed through retina, choroid, sclerotic, and conjunctiva. The stumps obtained by this procedure are extremely good, and they present an internal and external angular projection, on behalf of which it is claimed that they afford to an artificial eye a hold of peculiar excellence. I have practised this operation many times, and with the best results.

Early in last year, however, a strong, healthy, agricultural labourer came to St. George's Hospital, with complete staphyloma of his right cornea, the result of a wound from a thorn received while "hedging." The eye was lost as an organ of vision, and the protrusion was unsightly. I performed Mr. Critchett's operation, and obtained a stump which appeared to leave nothing to be desired. The man returned to his home, but came back in a month or six weeks with advanced sympathetic ophthalmia of his remaining eye. Enucleation of the stump was immediately practised; but the ophthalmia resisted all treatment, and ended in total loss of sight.

It is well known that the ordinary cause of sympathetic ophthalmia is irritation of a ciliary nerve. In this case it is possible that the irritation may have been due only to the traction of the cicatrix; but I think it more likely that a ciliary nerve was lacerated by one of the transfixing needles. Such an accident, although the chances are many that it will not happen, is one that cannot be guarded against; and I therefore set myself to contrive a substitute for Mr. Critchett's operation, one which should avoid all risk, and which should secure all its advantages.

For this purpose I now divide the conjunctiva, with

scissors, close to the corneal margin, and detach it from the subjacent parts nearly to the equator of the eyeball. I then take a squint hook with an eye in its point, carrying a fine silk thread, and by this means I pass a separate ligature under each of the recti muscles in succession. I tie each ligature around the tendon under which it lies, about a line from its insertion into the sclerotic, and then divide each tendon at its insertion, so that I can still command the muscles by the ligatures attached to them.

The next step is to remove, in the ordinary way, as much of the eyeball as circumstances may require. This done, I take a needle threaded with fine carbolized catgut, and pass it through the tendons of the superior and inferior recti, just behind the ligatures. The ligatures are then cut and removed, and the two tendons are united, in front of the gaping remains of the eyeball, by tying the catgut suture. The external and internal recti are then united in a similar manner by a second catgut suture. The ends of the catgut sutures are cut off as closely as possible, and the last step of the operation is to unite the edges of the conjunctival wound in a horizontal line, by three or four points of fine silk suture. In some cases the catgut threads cut their way out and are cast off, in others they remain and are absorbed; but this difference does not seem to affect the result. In any case, the coats of the eyeball, except the conjunctiva, sustain no other injury than a clean incision, and are not dragged upon in any way, either during or after the healing process. The recti muscles become firmly united in front of the stump, so as at once to form a protective cushion over its surface, and to act upon it advantageously as regards its movements. [A patient was in attendance on whom the operation had been performed three weeks previously, and the Fellows were able to judge for themselves of the quality and mobility of the stump.]

The operation requires a minute or two more for its performance than that introduced by Mr. Critchett, but this dose not constitute a serious disadvantage. It would be quite possible to dispense with the ligatures on the tendons;

but they give complete and immediate command of the severed muscles, and the time spent in tying them is probably redeemed by the convenience of their presence. The essential points which I wish to bring before the Society are the avoidance of injury to the ocular tunics, and the mobility conferred upon the stump by the slight advancement of the recti muscles.

THE

ETIOLOGY OF BRIGHT'S DISEASE

AND THE

PREALBUMINURIC STAGE.

BY

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COMMUNICATED BY

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(Received March 16th—Read April 14th, 1874.)

No classes of disease are more irresistible in their progress, or more hopeless in their prognosis when once fairly established, than those bearing the name of Bright; sooner or later, in one way or another, they invariably bring about the death of the patient. As we can do little or nothing towards their cure, except in the very earliest stages, it behoves us to find, if possible, some means for their prevention when threatening, or for their detention and arrest at their commencement.

I believe that the observations which I desire to discuss in the present paper take us one step, and that an important one, in this direction, and therefore they are, perhaps, worthy of record.

One of my earliest observations with the sphygmograph was that the pulse of acute Bright's disease closely resembles that which had previously been described and illustrated by the sphygmograph as occurring in chronic Bright's disease, or, more strictly speaking, with cirrhosis of the kidney. Both conditions were accompanied by a pulse of high tension —that is to say, one which usually required a considerable amount of pressure, and especially was distinguished by a prolongation or undue sustension of the tidal wave.

This observation has since been repeated and brought before the profession by Dr. Galabin in his thesis on "The Vascular Changes in Bright's Disease," but it only forms a small part of the truth of the matter.

When I observed this I also noticed that a somewhat similar, though not so well-marked, form of pulse occurred in other conditions, especially in the exanthems, erysipelas, and pregnancy, and that in these conditions albuminuria was particularly liable to occur. I therefore suspected that the vascular condition was the cause of the albuminuria, and not the albuminuria that of the vascular condition, as generally supposed. It is not, however, till lately that I have had the opportunity of testing and confirming my views.

The observations I have now to bring before you are briefly these:

1st. That previous to the commencement of any kidney change, or to the appearance of albumen in the urine, the first condition observable is high tension in the arterial system, due either to the presence of a noxious material in the blood, such as lead, alcohol, uric acid in gout, scarlatinal poison, or what not, which alters the relation between the blood and the tissues, and destroys their chemical affinity for each other; or else to a sudden chill causing contraction of the superficial vessels and congestion of the internal organs.

2ndly. If this condition of high tension be sufficiently severe, transudation of the characteristic crystalloids of the blood, notably hæmoglobine, occurs before albumen appears

in the urine, and they can be detected in that fluid by the guaiacum test for blood.

3rdly. If this condition be allowed to continue, albumen is subsequently found, and, if still unchecked or uncontrollable, Bright's disease in one of its forms ensues.

4thly. If checked before the albumen appears, or immediately after its appearance, by a brisk purge or other appropriate means, the condition is suddenly changed—the tension disappears from the pulse and the crystalloids from the urine.

Before proceeding further, it would be, perhaps, as well to give some particulars concerning the two indications to which I particularly desire to draw your attention. The characteristic features of a pulse of high tension may be enumerated as follows:

1. A pressure above one ounce, and sometimes as high as ten ounces, is employed to develop the pulse-tracing to its greatest extent.

2. The percussion wave is usually well marked and distinctly separated from the tidal.

3. The dicrotic wave is very small, and often scarcely perceptible; the vessels, however, are full during the diastolic period, and collapse slowly.

4. The tidal wave is prolonged and too much sustained. A similar result can be produced experimentally in a schema of the circulation by making the contraction of the ventricle laborious and prolonged, as it would necessarily be under these conditions of high tension. This feature is often indicated in the tracing merely by a slight rounding of the tidal wave, and any such tendency must be looked upon with suspicion; implicit confidence, however, cannot be placed in it, as a similar appearance is produced by degeneration of the arteries, but if due to this cause a very slight pressure is sufficient to extinguish the pulse, while a pulse of high tension generally requires a more considerable one.

The most constant of these indications is the prolongation of the tidal wave; any one or all of the other characters may under certain conditions be absent.

A few observations with regard to the manner in which I have employed the guaiacum test for blood are desirable. By a careful use of this test infinitesimal traces of blood, or rather its characteristic crystalloids, can be detected in urine which to the naked eye, to the microscope, to the spectroscope,[1] and even to the nitric acid test for albumen, affords no indication whatever of any abnormality. I am aware that this test is open to some fallacies; saliva, nasal mucus, and a salt of iodine in the urine (as happens when the patient is taking iodide of potassium), all cause a development of a blue colour with the tincture of guaiacum, some without and some after the addition of ozonic ether. But these fallacies, if a little care be used, can all be guarded against. Again, some believe that a faint development of blue cannot be held as conclusive of the presence of blood; but I feel sure when, after innumerable experiments with negative results, the appearance of a slight blue reaction is found invariably to herald the approach of, coexist with, and linger after, albuminuria, and at the same time, and as invariably, to be coincident with the signs of high vascular tension, all will agree with me in placing it in the first rank of our most valuable clinical tests.

My mode of procedure when applying it has been as follows:—One end of a small slip of white blotting-paper has been dipped in the urine and dried over the flame of a spirit lamp; by this means the dilute solution of the crystalloid is concentrated by evaporation; two drops of the

[1] I am indebted to Mr. Ashby, of Guy's Hospital, for many careful spectroscopic examinations. Mr. Ashby has devoted especial attention to the spectra obtained from the various constituents of the blood, and has published some of his observations in the 'Guy's Hospital Gazette' of Nov. 29th, 1873. Mr. Ashby failed to detect any sign of blood in the spectra obtained from urine which gave a slight reaction with the guaiacum test. In one case especially, in which the urine afterwards became loaded with blood, when the blue reaction was first discovered and was then well marked, Mr. Ashby could find no distinct indication in the spectrum. On the following day, when the reaction with the guaiacum test was intense, Mr. Ashby discovered sufficient indication in the spectrum to lead him to suspect the presence of blood. The urine was examined on each occasion by strong sunlight passing through a column of urine about eight inches deep.

tincture of guaiacum have then been dropped on the paper, and, after a minute or so allowed for the spirit to evaporate, a single drop of ozonic ether let fall in the centre of the guaiacum stain. Some time, perhaps a quarter of an hour, will elapse before the reaction becomes visible, especially if the reaction be slight; when it appears it is not permanent, it will begin to fade in a few hours, and will have disappeared in the course of a day or two.

One great advantage that the test appears to possess is the absence of all incumbrance to the physician; all that he requires to carry are a few slips of perfectly clean blotting-paper, one end of which he dips in the urine and dries, while on the other he can record the name of the patient; the test he can apply at home. Care must be taken that the urine be free from all contamination with saliva, &c., and that the patient is not taking any salt of iodine.

The guaiacum and ether must be fresh, and it is well to test both them and the blotting-paper employed, to guard against their contamination with anything that might give rise to a fallacious result. Especial care must be taken in selecting the blotting-paper; some papers give a slight but distinct reaction with the test, and I once nearly fell into many errors through this source of fallacy.

I may mention that on adding the ozonic ether an apparent bluing of the white paper ensues; this, however, is merely due to the moistening of the paper, and disappears entirely as it dries.[1]

[1] Since the present paper was written Dr. Stevenson discovered in a specimen of urine I took to him for examination that a far brighter and more perfect reaction can be obtained by applying the guaiacum to the fluid in the ordinary way than on the blotting-paper. From urine which gave by the above method but a faint reaction, requiring some practice to recognise it, a brilliant blue colouration was obtained by applying the test to the liquid. The mode of procedure is to place a drop or two of urine in a small test-tube; add one drop of the tincture of guaiacum and a few drops of ether; agitate and allow the ether to collect at the top, forming an upper layer of fluid. If hæmoglobine be present the ether carries up with it the blue colour that is produced, leaving the urine colourless below. In this method no error can arise from the blotting-paper, which is no longer required, and was a frequent source of fallacy.

I have applied the test to urines of all descriptions—to the pale urine of hysteria, to intensely high-coloured urine, to that of phthisis and other wasting diseases, to urine containing bile, sugar, excess of uric acid, lithates, phosphates, and excess of mucus, but in none was any reaction produced.

Finally, the class of cases for which this test is available appears disappointingly small, though, perhaps, on this account the indications obtained by it are still more valuable.

The reaction can be obtained only in the earliest stage of either the acute or chronic form of Bright's disease, usually before the albumen appears or when it is present in only small quantities. It does not occur in chronic albuminuria, except when blood is also present in the urine; when this happens the guaiacum test gives by far the earliest indications of its presence that can be obtained. Even in those cases in which it precedes the appearance of albumen it fades when the albumen becomes copious, and appears again as it diminishes or after it has disappeared. In cases of albuminuria produced by intense fever and due to venous congestion, as in enteric fever, pneumonia, and sometimes in the febrile stage of scarlatina, when the fever is intense and the albuminuria only slight, no reaction showing transudation of the crystalloids can be obtained. It thus forms a distinction between some of the various forms of albuminuria which possesses the deepest interest, and perhaps affords a key to the mechanism of albuminuria and the method of its production. These matters, however, will be referred to hereafter.

The series of cases to which I especially desire to draw your attention this evening are all of one type. Their characteristic features are these:—

They all occur in patients recovering from scarlet fever. The symptoms in question may arise at any time during desquamation, namely, from the defervescence of the fever to six weeks or even longer afterwards. Their almost universal forerunner and probable cause is

constipation; another but much less frequent cause is a chill; in the cases to be considered constipation is almost invariably present. One day passed without an action of the bowels is sufficient to give rise to the symptoms in question. The immediate result of this constipation appears to be the retention of a quantity of effete material in the blood, which, by disturbing the relation between that fluid and the tissues, interferes with the capillary circulation and produces a condition of high tension throughout the system. The condition is indicated by the following symptoms:—By the signs of high arterial tension afforded by the sphygmograph which have already been enumerated; by a more or less marked blue reaction if the guaiacum test be applied to the urine as above described. In this first stage, if the tension be not very high, no albumen may be found in the urine; if the symptoms are not immediately relieved, on the following day the blue is either brighter or albumen appears in the urine either in small or large quantities; if it be still neglected or prove intractable, the more severe signs of kidney disease occur, such as bloody and scanty urine, dropsy, uræmia, &c. If, however, these symptoms are detected and a sharp purgative be administered, they fade as rapidly as they came; the blue reaction disappears from the urine and the high tension from the pulse. The albuminuria is variable in its intensity; in some only the prealbuminuric stage occurs, the further development of the disease being arrested; in other cases, which appear to be due to chill rather than to constipation, no reaction with the guaiacum test is obtained, the albumen, however, being very copious; again, in others the blue reaction is obtained both at the commencement and after the disappearance of the albumen from the urine, though not while it is abundant; in fact, a large quantity of albumen or chronic albuminuria alike appear incompatible with the transudation of the crystalloids, though high tension is still exhibited in the sphygmographic tracings obtained in these cases.

The relation of albuminuria to rheumatism in scarlatinal patients affords another most interesting and important

problem for solution; both appear frequently to be produced by the same cause, namely, constipation, yet the two appear somewhat antagonistic, for the tendency of a rheumatic patient to albuminuria often appears much less than in other cases, yet the two conditions occasionally coexist in the same person.

To return to the cases, however, these symptoms are occasionally, but not invariably, accompanied by a slight increase of temperature; this varies from 99° to 100° Fahr., and is highest in the morning. The pulse is hard and sustained. The signs of high vascular tension pointed out by Dr. Sibson are present in well-marked cases, namely, a forcible and sustained apex beat, prolongation of the first sound of the heart, and a reduplication of this sound near the apex over the septum, while at the base the first sound is dull and indistinct, the second loud and clacking. There are often no constitutional symptoms, the appetite being good, and no pain being felt either in the back or in the head; in fact, these patients often express themselves as feeling well. But in some cases lumbar pain is complained of, in others there is loss of appetite and sometimes vomiting. In many cases slight œdema about the lower extremities can be detected by careful examination.

Unfortunately the amount of urine passed in twenty-four hours has not been measured in the cases quoted, but in other similar cases this observation has since been made. In these it has been found that the amount of urine secreted is invariably diminished when the tension of the pulse is increased or albumen or crystalloids are present; the amount of urea excreted by the kidneys under these conditions being very markedly diminished. Microscopical examination of this secretion yields no definite result, for in the early stages neither blood-cells nor desquamating swollen renal epithelium-cells are found; it is only when the condition has lasted for some few days that the latter can be discovered. I believe, therefore, that the term acute desquamative nephritis does not convey the true impression of the case, for the shedding of the epithelium only occurs as a secondary

condition; it is due to the increased proliferation of epithelium-cells from the increased blood supply. The further discussion of this matter I must defer till a later period. In the mean time I propose briefly to refer to a few illustrative cases and exhibit the sphygmographic tracings obtained from them; unfortunately I cannot at the same time show the amount of blue reaction by the guaiacum test that occurred in each, as the blue does not remain permanent, but fades in a few days.

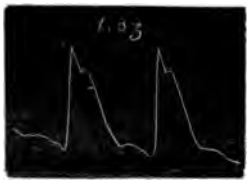
The cases of scarlet fever referred to in this paper were all admitted into the London Fever Hospital under the care of Dr. Broadbent, by whose kind permission I make use of them.

It will be unnecessary to quote any of those severe cases of renal disease following scarlatina whose well-known symptoms are scanty and bloody urine, and early dropsy, which run the usual course of acute Bright's disease, and die either in the first, second, or third stage. With these all are familiar; the cases on which this paper is founded illustrate the commencement of these by symptoms which I believe are usually overlooked; some of the present cases, if not arrested, would probably have developed to the same extent, while others would have passed off unnoticed both by patient and physician.

The pulse of scarlatina is one of high tension from the commencement; even in the most severe cases it never becomes markedly dicrotic, though in some conditions the tension falls greatly during diastole, so that the diastolic portion of the pulse-trace becomes an almost horizontal line, yet the dicrotic wave is not developed. Fig. I and Fig. II, 1, are examples of this form of pulse occurring in very severe cases.

No. I.—Fig. I was obtained from a lad, aged seventeen, who had an extremely severe attack of scarlatina complicated by pericarditis and pneumonia, from which he ultimately recovered. During the greater part of his fever his urine was albuminous and at one time bloody.

FIG. I.



Eighth day of fever. Intense fever. Urine albuminous.

The woodcut illustrates his pulse when most nearly approaching dicrotism. The prolongation of the tidal wave is well marked, and it shows considerable tension.

No. II.—Though the urine never became actually albuminous in the following case, the condition which immediately precedes it was once present.

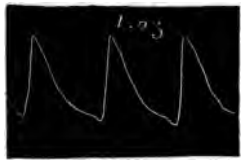
A. D—, a clerk, æt. 19, was admitted into the London Fever Hospital on November 26th, 1873, suffering from scarlatina anginosa. Five days previously he was exposed to contagion. The day of admission was the third of his fever and the second of the rash. His attack was a severe one; his temperature was very high, he had great œdema of his uvula, and extensive sloughing of the left tonsil and anterior pillar of the fauces, with a tongue dry and brown in the centre. Fig. II, 1, represents his pulse on the fifth day of his illness; the urine was not albuminous, and the tidal wave is not sustained (in Fig. I an exactly opposite condition exists). On this day there was the greatest amount of dicrotism that was ever present, and yet this wave is very small.

On November 29th, the sixth day of his fever and the most critical of his illness, with his temperature at 105° and the slough in his throat increasing, his pulse appeared to be losing power (Fig. II, 2), the heart was becoming exhausted, and owing to its enfeebled contraction the percussion wave has disappeared, the upstroke being rather sloping; a similar condition in a continued fever would be accompanied by hyper-dicrotism, but in scarlet, the pulse, which is non-dicrotic throughout, is especially so in this condition. Under the influence of free stimulation and good nourish-

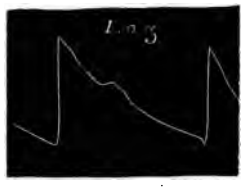
FIG. II.

1. Intense fever. Urine normal. Nov. 28th, 5th day.

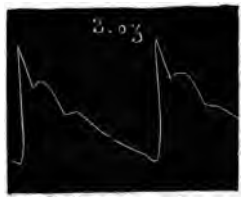
2. Intense fever. Urine normal. Nov. 29th, 6th day.



3. Convalescent. Urine normal. Dec. 6th, 13th day.



4. No albumen. Blue with guaiacum test. Dec. 20th, 27th day.



5. No albumen. Blue with guaiacum test. Dec. 21st, 28th day.

ment he passed safely through the crisis, and the following day his temperature had fallen and his pulse regained power. His urine was free from blood and albumen during the whole of his fever.

On December 1st, the eighth day of his illness, he had an

attack of rheumatism, his urine on this day containing an excess of uric acid; this excess is almost invariably present in cases of scarlatina at about this period, and, perhaps, accounts for the coincident rheumatic tendency. His bowels had been confined for one day when the rheumatism appeared, and it is probably to the arrested excretion of effete material by the bowel that the rheumatism was due; his pulse-tracing exhibited no increase of tension coexistent with the attack of rheumatism.[1] During the next five days, the bowels being purged, the rheumatism quickly subsided, the uric acid in the urine decreased, the temperature became normal, and the pulse assumed a healthy form, which it maintained during the greater part of his convalescence. Fig. II, 3, is an example of this type of pulse obtained on the thirteenth day of his illness.

We now come to a series of phenomena to which I particularly desire to call attention. During the twelve days succeeding that on which the last tracing was obtained his bowels continued freely open, convalescence proceeded satisfactorily, and his urine and pulse remained normal; but on the twenty-sixth day of his illness his bowels were rather constipated and his pulse, which was irregular, showed a decided increase of tension; his urine, however, gave no abnormal reaction.

On the following day he had no action of the bowels what-

[1] As mentioned above, the same condition, namely, slight constipation, in similar cases is alone required to produce albuminuria, and a question of extreme interest presents itself for solution by the physiologist, namely—What chemical conditions determine whether this transient constipation will produce rheumatism or albuminuria in a given case? That the two are closely allied seems certain; yet in two otherwise exactly similar patients, why do we find in one increased tension and albuminuria, in the other no increase of tension and rheumatism? It is possible that in the albuminuric patient the chemical changes are not sufficiently rapid, and effete albuminous and therefore colloid materials are present which impede the capillary circulation, and when increased in amount by constipation produce high arterial tension and albuminuria; while in the rheumatic patient the changes have been more rapid, the colloid being changed into the crystalloid, uric acid, and though the capillary circulation is less impeded, its presence in an excessive degree gives rise to an attack of rheumatism.

ever, and the tension of the pulse became greatly increased (Fig. II, 4), three ounces of pressure being now required to develop it, whereas one ounce was previously sufficient; the tidal wave, moreover, was too much sustained. On applying the guaiacum test to the urine a distinct blue reaction was obtained, showing transudation of the hæmoglobine and perhaps other crystalloids of the blood, though the most careful testing failed to discover any trace of albumen. On the following day, after a purge, the bowels were relieved, but not freely so; the blood pressure was immediately reduced, two ounces sufficing to develop the pulse (Fig. II, 5), but the tidal wave continued sustained. The urine still gave a blue reaction with the guaiacum test, though fainter than before; no trace of albumen could be discovered by the most delicate testing. The purge being repeated, the bowels were freely open on the succeeding day, the tension became much less, the pulse wave being larger and the tidal less sustained; the pulse had again become irregular, owing to the heart not having yet adapted its action to the changed vascular condition. The urine now gave no reaction with the guaiacum test, and it remained free from albumen. The patient left the hospital on the following day, but appeared a fortnight after to report himself; both the pulse and urine were then normal. During the time when this increased tension existed he complained of no constitutional symptoms whatever, and considered himself in his usual health. The case possesses much interest as showing the earliest commencement of a condition that might have developed into Bright's disease, its detection and immediate arrest.

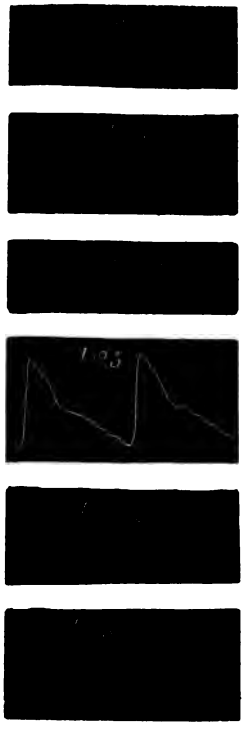
No. III.—The next case I desire to bring before you affords a typical illustration of the class to which it belongs.

G. A—, a lad in a city warehouse, was admitted into the London Fever Hospital January 25th, 1874, suffering from scarlatina, on the second day of his fever. His attack was not a severe one. He had a profuse rash and free desquamation; he became convalescent without any complication. During the whole of his convalescence he had a tendency to

constipation, which was treated by frequent purgatives. On the twentieth day of his illness, his bowels having been confined for one day, his urine was found to be albuminous, and a blue reaction was obtained with the guaiacum test.

On the following day, his bowels being still confined, his urine was highly albuminous, while the reaction with the guaiacum test was diminished. Fig. III, 1, was obtained on

FIG. III.



1. Albumen. Blue with guaiacum test. Feb. 14th, 21st day.

2. No albumen. Blue with guaiacum test. Feb. 17th, 24th day.

3. Much albumen. No blue. *Before pack.* Feb. 22nd, 29th day.

4. A trace of albumen. No blue. *After pack.* Feb. 22nd, 29th day.

5. Much albumen. No blue. *Out of bed.* May 15th.

6. No albumen. No blue. *In bed.* May 15th.

this day; it shows a condition of high tension and contracted arteries; three ounces of pressure was required. Although he had this well-marked albuminuria, he suffered from no constitutional symptoms whatever; he expressed himself as feeling

in every way quite well. He had no nausea or pain in the loins. On the following day, the twenty-second of his illness, having been well purged, his urine contained only a faint trace of albumen, which was discovered with difficulty, while the blue reaction was now more distinct. The pulse-tracing still indicated the same high tension as on the previous day. The blue reaction lasted for several days longer; it existed altogether for nine days, albumen being present only on the first three, and on only one in any large amount. During the remainder of the time the pulse exhibited a gradual diminution of tension and return to the normal form. Fig. III, 2, obtained three days after the last, shows the arteries relaxing and the pressure decreased.

In this case the ordinary physical signs of high tension were present; the heart's impulse was forcible and sustained, the first sound was dull and prolonged, near the apex reduplicated, but at the base distant and indistinct, while the second was loud and clacking at this spot. There was slight subconjunctival œdema, and by firm pressure slight pitting could be obtained over the tibiæ. The ophthalmoscope revealed nothing abnormal. His appetite was good throughout. His morning temperature ranged between 99° and 100°, while in the evening his temperature fell below normal. The day his temperature became normal the blue disappeared coincidently from his urine and the tension from his pulse. He was allowed to get up on the following day; he did so, however, without wearing flannels and received a chill; his bowels were at the same time confined. On the next day, the twenty-ninth of his illness, his urine was found to be loaded with albumen, but no reaction was obtained with the guaiacum test; his pulse-tracing again indicated high tension and very contracted arteries (Fig. III, 3). He was placed in a hot pack for two hours, and profuse perspiration was induced; by this means his condition was completely changed. The next urine secreted contained only a trace of albumen, the blue reaction was still absent, and his pulse-tracing demonstrated a relaxation of his arteries, a great reduction of tension, and a normal condition of pulse (Fig.

III, 4). His bowels, however, still remained confined, and although on the following day his urine was normal the pulse indicated rather too much tension. On the succeeding day, after a free purge, his pulse became perfectly natural, the tidal wave being scarcely perceptible, and the urine and temperature were also normal. On four occasions after this his urine became albuminous; each time the albumen was copious, and no reaction was obtained with the guaiacum test. His urine during this period was measured and found to be deficient in quantity; on one occasion it was as low as 22 oz., and it never exceeded 48 oz. It was noticed at this time that when up and about he invariably suffered from cold extremities; this symptom is very frequently present in scarlatinal convalescents who are liable to albuminuria; they would be popularly described as suffering from "bad circulation," and such an expression would be scientifically correct if qualified with the word "capillary." The cold extremities are due, I believe, to "bad *capillary* circulation," the poisoned blood moving slowly through the capillaries from diminution of capillary power or chemico-vital attraction, hence the heart has increased work and high tension is produced. With this symptom unchanged he went out of the hospital on March 27th, having been ill about nine weeks. He continued under observation, and albumen reappeared in the urine; as it remained persistent he was readmitted on April 24th, it being then upwards of three months since he was taken ill.

The scarlatinal poison was shown to be still existent in a most striking manner; on his returning to his house of business, about a week previously, he communicated the disease to another lad who shared his sleeping room. He still suffered from cold extremities, and the interesting discovery was now made that while keeping his bed his urine was perfectly normal, but that immediately he left it his urine became highly albuminous. This change was instantaneous, half an hour in either condition sufficing to alter the character of the urine. Corresponding to this altered secretion there existed the changed condition of circulation

on which it depended. Fig. III, 5, illustrates his pulse while out of bed, his urine being albuminous; while Fig. III, 6, represents his pulse on the same day, three quarters of an hour after taking to his bed, the urine being normal; at the same time it was ascertained by volumetric analysis, albuminuria being produced, that a large diminution in the excretion of urea occurred from that which took place in a corresponding period of time while the patient was in bed and the urine normal, although the diet on each occasion was exactly similar. This fact is easily accounted for by the diminished tissue changes which take place when the capillary circulation is retarded.

These symptoms were not overcome till he was clothed in thick flannel from head to foot, and he left the hospital apparently cured five calendar months after his first admission.

No. IV.—The following case was one in which albuminuria never actually occurred, though the prealbuminuric condition of high tension and transudation of crystalloids was at one time present.

E. W—, a butler, æt. 29, was admitted into the London Fever Hospital on the second day of his fever, suffering from a severe attack of scarlatina anginosa. His throat presented patches of exudation almost diphtheritic in appearance, while his urine during the fever was slightly albuminous, though it gave no reaction with the guaiacum test. During his convalescence he had several slight attacks of rheumatism, and his temperature kept up, varying from 99° to 101° for thirty-four days, though without any apparent cause. His appetite was good and his bowels regular, with rather a tendency to be relaxed. The first tracing on the card was taken on the twentieth day of his illness, his bowels being open, urine normal, and pulse-tracing perfectly natural. On the twenty-seventh day, however, his bowels having been slightly confined, his pulse shows a great increase of tension; in appearance it exactly resembles one of chronic Bright's disease. The urine,

FIG. IV.

1. Normal. Feb. 12th, 20th day.

2. No albumen. Blue with guaiacum test. Feb. 19th, 27th day.

3. No albumen. No blue. Feb. 20th, 28th day.

4. Normal. Feb. 21st, 29th day.

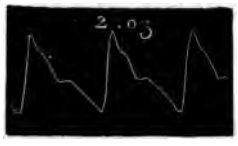
though containing no albumen, gave a well-marked reaction with the guaiacum test. The following day, after a purge, the tension was considerably diminished (Fig. IV, 3), while the crystalloids could no longer be detected in the urine. On the following day both temperature and pulse (Fig. IV, 4) were normal. This condition never recurred in this patient.

No. V.—C. W—, a housemaid, æt. 19, had been nursing scarlet-fever patients; she believed this to be her second attack. Her fever was a very mild one. She was admitted on the second day of her illness. She had very little rash and merely a slight reddening of her fauces. Her bowels had a tendeney to constipation throughout. The tracing obtained on the day of her admission indicates contracted arteries and a firm pulse, but the tidal wave was not

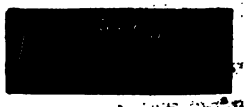
FIG. V.

1. Normal. Nov. 21st, 2nd day.

2. Albumen. Blue with guaiacum test. Nov. 27th, 8th day.



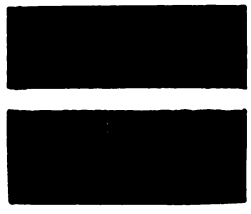
3. No albumen. Very faint blue. Nov. 29th, 10th day.



prolonged. Her urine was now normal. Three days after this she suffered much from earache and otorrhœa. Her bowels were confined, but the urine was normal. On the eighth day of her illness, her bowels not having been open for two days, her urine was found to be faintly albuminous, while it gave a distinct reaction with the guaiacum test. Her pulse was one of well-marked high tension. She complained of pain in the small of her back and headache. She had subconjunctival œdema, the face looked puffy, and her legs were slightly œdematous. On the following day, her bowels having acted once only, her symptoms were but partially relieved. The urine was still faintly albuminous, though the blue reaction was very much less than on the previous day. The tracing obtained on this day showed a decided diminution of tension. On the next day, her bowels having been further relieved, her condition was altogether improved. The pain had disappeared from her back. Her temperature became normal for the first time, while her urine contained no albumen, and gave but a very faint reaction with the guaiacum test. The tension of her arterial system was diminished, her pulse-tracing being normal, though still firm, with somewhat contracted vessels (Fig. V, 3). Her legs continued slightly œdematous for three or four days after this, but her urine never again became albuminous. It is interesting to notice that in this case,

although the albumen was very scanty throughout, the constitutional symptoms were well marked; while in the lad previously mentioned, whose urine was highly albuminous and the albuminuria frequently recurring, no constitutional symptoms were ever present.

FIG. VI.



1. Albumen, and bright blue with guaiacum test. Oct. 25th, 24th day.

2. No albumen. Blue less intense. Oct. 30th, 29th day.

No. VI.—The two tracings in the above woodcut afford good examples of the condition of high tension accompanying albuminuria in a child of five, during his convalescence from a mild attack of scarlatina. On the twenty-fourth day of his illness, when the first tracing was obtained (Fig. VI, 1), the albuminuria had certainly existed three days, and it may have been longer; the tension had, moreover, been relieved by a sharp purge on the previous day.

The second tracing was obtained on the day that the albumen disappeared, the blue reaction still remaining, though less marked. The tension of the pulse has greatly diminished, and the muscular coat of the arteries has relaxed, so that the pulse is larger; a day or two afterwards the blue completely disappeared. In this case transudation of the crystalloids of the blood occurred throughout, and continued for several days after the disappearance of albumen from the urine.

No. VII.—The following case has been substituted since the reading of this paper for one previously inserted, in order especially to give a well-marked example of the

blue reaction affording the first indication obtained from a urine that afterwards became distinctly bloody.

FIG. VII.

1. Much albumen. Bright blue. May 30th, 27th day.

2. Faintly albuminous. Bright blue. June 6th, 34th day.

W. B—, a boy, æt. 13, was admitted into the London Fever Hospital on May 5th, 1874, suffering from a severe attack of scarlatina; his temperature was very high, ranging from 104° to 105° up to the eighth day of his fever; his throat was severely ulcerated and sloughy. After the defervescence of his fever his temperature kept high during his convalescence, varying from 100° to 101°; he, however, appeared well in himself, and there was no apparent cause for the high temperature. On the twenty-third day of his illness his bowels were confined, and his urine on this and the following day gave a blue reaction with the guaiacum test. After a sharp purge his urine, on the twenty-fifth day, was again normal; on the evening of this day he had a bath, and got up the following day; the urine secreted after this was albuminous, and again gave the blue reaction; on this day, the twenty-sixth of his illness and the first of leaving his bed, the urine secreted in the twenty-four hours measured only 15 oz. A pulse-tracing obtained on the following day (Fig. VII, 1) shows a condition of cardiac excitement with exaggerated percussion and tidal waves, a form of pulse which is often seen in young subjects suffering from acute Bright's disease. His temperature began to rise rapidly on this day, reaching 102° in the evening, and the glands at the right angle of the jaw became inflamed and swollen; his urine was albuminous, and the blue reaction very bright.

On the following day (the twenty-eighth of his illness) his temperature had risen to 103·6°, while his urine was albuminous and gave a very bright blue reaction; on this day it was distinctly bloody, and many blood-corpuscles were seen by aid of the microscope. Early the next morning a sharp purgative was administered, which produced nine actions of the bowels, and reduced the temperature to 98·6°; the pulse also fell from 116 to 72 per minute; the blood disappeared from the urine; the blue reaction remained, but only a small amount of albumen was discoverable. Three days after this the same symptoms were repeated. Glandular swelling occurred at the opposite angle of the jaw, and the temperature rose to 104·5°, the urine again becoming bloody; but the purge being repeated, the temperature fell the next day and the condition of the urine improved, though not so much as on the first occasion.

After three days his urine was only faintly albuminous, but the blue reaction was still well marked; this condition persisted for a long time, up till the fifty-fourth day of his illness, that is, thirty days since the appearance of the blue. The albumen was variable in amount during this period, and on one or two occasions disappeared entirely; for three days the blue reaction also disappeared, and again returned. While in this condition the urine was examined by the spectroscope, but no characteristic blood spectrum was obtained. The pulse, of which Fig. VII, 2, is an example, retained the same character throughout; it is typically one of high tension. He had no œdema nor any constitutional symptoms, his bowels were kept freely purged. On the fifty-third day he was placed in a hot pack, and copious perspiration was induced; on the following day his urine was perfectly normal, and continued so for the next ten days, during which he remained under observation.

With regard to this case it is highly interesting to observe that the chief cause of his symptoms appears to have been a *chill*, and although purgation very greatly relieved them they were not removed till his skin was brought thoroughly into action by the hot pack.

From these few typical cases I think some important deductions may be drawn; doubtless they are already foreseen. I believe it is now generally admitted that the renal disease following scarlatina is exactly similar in its nature and progress to that usually known as the inflammatory form of Bright's disease; it will probably be agreed, moreover, that the cases I have quoted in the present paper indicate the earliest symptoms of this disease, and it will also be allowed that some, if not all of these, might have developed under unfavorable circumstances into the more severe condition. We have, then, during the convalescence of scarlatina, an opportunity afforded us for watching the mode of origin and first indications of the ordinary so-called "inflammatory Bright's disease."

But can the term "*inflammatory*" be fairly applied to this disease? I take it that, correctly speaking, it cannot; it does not describe the true condition, and it must convey a false impression of its pathology. It is not an actual inflammation of the kidney that gives rise to the symptoms produced. It is a general blood poison, by which the kidney is not alone affected, but it suffers in connection with other organs, notably those of excretion. This view is not a new one; a lecture by Dr. Walshe is published in the 'Lancet' of 1849, entitled "Bright's Disease not a Renal Disease, but primarily a Blood Disease," in which that accomplished physician arrives at this conclusion, though from arguments of a different nature; he deduces his theory from the clinical history of these cases, and takes a case of renal disease following scarlatina as an example.

Drs. Fenwick and Wilson Fox have both demonstrated that a similar condition to that which exists in the kidney exists also in the stomach and duodenum; it probably exists also throughout the intestinal tract. They have found inflammatory Bright's disease associated with a corresponding affection of these organs, namely, increased proliferation of the cells lining the follicles, producing distension of the follicles with cells and granular matter, similar to the affection of the kidney-tubules, while cirrhosis of the kidney they have found accompanied by intertubular changes.

We may moreover anticipate, reasoning by analogy and from experience, that the skin is affected in a similar manner, namely, that the sweat-glands become blocked from a similar proliferation of cells, and are unable to act, thus producing the dryness and roughness of the skin with which all are so familiar in Bright's disease. This, however, I have never substantiated by observation, nor have I seen it described; if it should be so we have all the organs of excretion simultaneously affected and in a similar manner; then, why should the kidney give its name to the disease except because its symptoms are most urgent, and therefore cause the others to be overlooked?

Again, while the condition is not solely confined to the kidney, neither is it inflammatory, for what appears to be the true clinical and pathological condition? A patient having a quantity of effete or poisonous matter in his system, that he is excreting as rapidly as possible, has the function of one of his excretory organs suddenly or gradually arrested—it may be his bowels or it may be his skin. Coincident with this we notice a sudden or gradual increase of tension produced in his vascular system generally; the poisoned blood loses its affinity for the tissues, it is already surcharged with matter to be excreted, and cannot take up much more; the force of chemical attraction is diminished, if not lost; the remaining organs of excretion, previously heavily taxed, have now to do the work of the third, and, whether it be that the bowels are confined or the skin chilled, extra duty falls in each case upon the kidney. This organ, therefore, receives an increased blood supply, and that under a higher pressure than usual. But surely this condition hardly amounts to an inflammation? A muscle in active exercise and the uterus at the menstrual epoch both have an increased blood supply, but neither could be described as "inflamed." The increased tension, moreover, is not confined to the kidney, but is present throughout the arterial system.

The first indication obtained from the kidney of this increased blood supply and high pressure is the transudation of some of the crystalloids of the blood, indicated by a

blue reaction, more or less marked, obtained with the guaiacum test. If this condition be allowed to continue we find that albumen next appears in the urine in greater or less amount; the crystalloids may continue to transude, or as the albumen becomes copious they may disappear. The fact that these two constituents of the blood are not always present in the urine simultaneously appears to indicate that it is only under a certain degree of pressure that the crystalloids pass through the walls of the vessels, and that when the pressure is increased they cease to do so, but the colloid albumen now appears in the urine; if the pressure becomes still greater, rupture of minute vessels occurs, and blood is poured out into the kidney-tubules.

The correctness of this view of the relative diffusibility of these constituents of the blood under varying pressures might, doubtless, be readily tested by experiment; it may also be shown in this manner that albumen is capable of diffusion through the capillary walls without any pathological change occurring in them. This, however, has not yet been attempted.

During this condition of high arterial tension, whether crystalloids or albumen appear in the urine, the quantity of this fluid I have found to be invariably reduced.

So far we have traced the kidney to a condition of congestion under high pressure and seen its consequences; arguing from our knowledge of the effect of increased blood supply to any part, the next step in the process is evident—it will be an increased growth of the tissues supplied, namely, the epithelium of the tubules, and this exactly agrees with what pathologists have described. New cells are too rapidly formed, old ones too rapidly shed; these cells, moreover, growing like vegetables forced under conditions most favorable for their growth, are increased in size as well as in number; plugging of the tubules is therefore readily accounted for by either exudation of fibrine or distension with swollen cells.

I need pursue the subject no further; it is now becoming generally admitted that in the so-called "inflammatory" form the condition of the kidney gradually changes from the large

red to the white, and then to the granular and contracting form. Yet in no stage does a process of a truly inflammatory nature occur. From the condition of high tension in the vascular system that precedes and accompanies these kidney changes it will be readily judged how necessary an accompaniment must be the hypertrophy of the heart and arteries. I have attempted to prove that this high pressure in the vascular system is not the result of kidney disease, but of a primary blood poison.; and I believe that the fact of increased pressure demonstrated to exist in the capillaries, by exudation through their walls of the crystalloids, negatives Dr. Johnson's view of the production of hypertrophy of the heart by the stopcock action of the arterioles, for if this existed the pressure in the capillaries would not be increased;[1] I should imagine, moreover, that the effort of the muscular coat of the arterioles to prevent over-distension by the increased pressure would be sufficient to account for their hypertrophy, while the increased work of the heart to overcome the increased pressure in the vessels would be amply sufficient reason for a similar change in that organ.

Lastly, with regard to all cases of acute Bright's disease, what I have said of those of scarlatinal origin appears to hold good. For while in those of scarlatinal origin we have an evident blood poison producing high tension, yet in all cases of Bright's disease a similar cause is undeniably present; it may be produced by a severe chill causing arrest of the action of the skin, general visceral engorgement, and increase of vascular tension from contraction of the cutaneous vessels, similar to that produced by a cold bath, a condition most readily demonstrated by the sphygmograph (such, probably, were those cases lately brought forward by Dr. Johnson in his paper on "Temporary Albuminuria after Cold Bathing," read before the Clinical Society of London);

[1] Experiments have been performed by Dr. Lauder Brunton and the author (to be reported elsewhere), in which both the blue reaction with the guaiacum test and albumen were discovered in the urine of a dog, in whom high arterial tension had been produced by ligation of the abdominal aorta below the renal vessels.

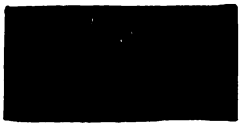
or an actual blood poison may exist, as in pregnancy, erysipelas or measles (both of them acute diseases accompanied by a pulse of high pressure like scarlatina), and in poisoning by uric acid in gout, by lead, by alcohol, or any other of the usual causes; alike under all these circumstances a predisposing condition of high pressure in the vascular system is demonstrated by the sphygmograph before any symptom of Bright's disease presents itself. Examples of a few of these are afforded by the typical tracings reproduced in Fig. VIII, the first four of which are chronic, the last two, acute conditions.

Pregnancy is well known to be frequently associated with albuminuria; it is, moreover, always mentioned among the causes of Bright's disease, while puerperal convulsions are considered by many to be of a uræmic nature, and are usually found to be accompanied by albumen in the urine. This condition has been ably discussed by Dr. Braxton Hicks in his paper "On the Pathology of Puerperal Eclampsia," published in the eighth volume of the Obstetrical Society's 'Transactions.' After demonstrating the frequent existence of albuminuria with eclampsia, he admits that the relation of one to the other has not been arrived at; he cannot say whether the eclampsia is caused by or is a cause of the renal disease, or whether both are due to one cause. The problem is, however, very clearly solved by the sphygmograph. I have found, in a large proportion of the pregnant women whom I have examined, high tension existing in the pulse (Fig. IX, 1 and 2). In none was the pulse that of low tension or dicrotic. This condition of high tension is especially confirmed by the hypertrophy of the heart which accompanies pregnancy. During the first stage of labour the pulse (Fig. IX, 3) is invariably one of high tension. This condition of tension decreases gradually after labour; it requires from a week to three or more to assume the normal form. Fig. IX, 3, 4, and 5, show the gradations of tension in one patient from the first stage of labour on the first and on the twelfth days, that obtained on the last day being almost normal.

FIG. VIII.

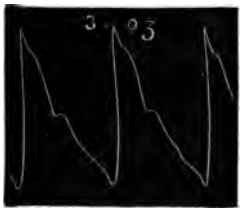
Pulses of high tension from blood poison. Urine not albuminous, but likely to become so.

1. *Gout.* Æt. 34.



2. *Chronic dyspepsia.* Malassimilation. Æt. 55.

3. *Lead.* Æt. 48.



4. *Alcohol.* Æt. 58. Delirium tremens.

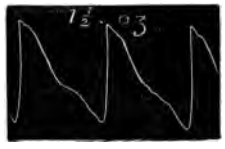
5. *Erysipelas.* Æt. 43. Temp. 104·8°.

6. *Measles.* Æt. 14. Convalescent.

We have already seen the relation borne by high tension to kidney disease; we have, therefore, an evident predisposition to this disease, demonstrated by the sphygmograph, during pregnancy. Unfortunately I was unable to procure the urine for examination in these cases; I believe that a

FIG. IX.

1. Pregnant 8½ months. Æt. 24.

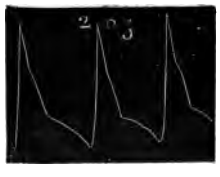


2. Pregnant 7 months. Æt. 34.

3. First stage of labour. Æt. 30.

4. First day after.

5. Twelve days after.



reaction with the guaiacum test might often be obtained in such cases before albumen appears. The reason for this high tension is evident. The blood of the mother is overcharged with effete material, for she has to discharge the excrementitious matters of the fœtus by her own excretory organs; her blood is therefore in a measure poisoned, and like a scarlatinal convalescent does not bear its normal relation to the tissues, while the organs of excretion have to do increased work, and that under increased pressure; should, then, one of them be interfered with, as by constipation

or a chill, albuminuria appears a very probable result. This, if not soon relieved by a purge or other appropriate means, would readily develop into the more severe forms of Bright's disease.

Finally, the condition best described as cirrhosis of the kidney I believe to have a similar origin to those already mentioned. The condition of increased vascular tension from blood poison, especially that of alcohol, gout, or lead, still exists, although in a more chronic form (*vide* Fig. VIII, 1, 2, 3, and 4).

This description of blood poisoning having generally commenced insidiously, the tension has been very gradually increased, and, although a very high degree of tension is eventually arrived at, the increase has been so gradual, and the process has extended over so long a period, frequently many years, that the vessels generally, including the arterioles and capillaries, have undergone a corresponding thickening. This change in the vessels probably satisfactorily answers the question, Why is albuminuria so frequently absent in cirrhosis of the kidney? It also accounts for the crystalloids not transuding and no reaction being obtainable with the guaiacum test. This condition, namely, blood poison, high tension, and arterial degeneration, may occur without corresponding kidney change if either by the occupation, habits, or constitution of the patient, the skin or bowels do increased work and relieve the kidneys from congestion; furnace men and others accustomed to copious perspiration occasionally afford instances of this. These cases may be included under the term arterio-capillary-fibrosis.

As long as no existing cause, such as chill, producing visceral engorgement and arrested action of the skin, or any great interference with the bowels, occurs to give rise to the symptoms of the acute disease, the prolonged condition of high tension combined with a chronic and not intense congestion of the excretory organs would produce cirrhosis. For Sir William Jenner, in his paper communicated to this Society on "Congestion of the Heart," published in the 43rd

volume of its 'Transactions,' has described a very similar change, namely, induration and contraction of organs, especially the kidneys, as due to that cause. I believe that the chronic condition of increased blood supply produces increased growth of the intertubular connective tissue, and if still continued this must result in contraction and induration; atrophy of the tubules from constant pressure would now ensue. This sequence of kidney changes agrees with that described by most observers.

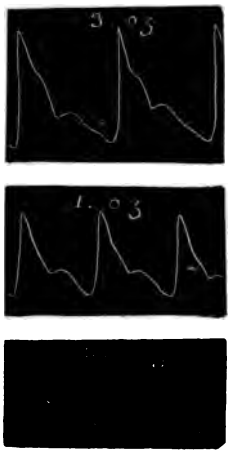
In confirmation of this view of the commencement of these changes I have frequently observed a condition of high tension in the pulse in cases that have subsequently developed albuminuria and symptoms of cirrhosis of the kidney, and in one or two out of a small number of observations I have obtained a blue reaction with the guaiacum test in cases where albumen was detected in the faintest possible traces, and even when it was absent. Of these I may quote one; it is that of a man of gouty family history and a free drinker, who had suffered from frequent "bilious attacks" and loss of appetite in the morning for some months before he came under observation; he was admitted into the Fever Hospital October 5th, 1873, suffering from enteric fever. Though his fever was not severe, his urine was loaded with albumen from the time of his admission; his pulse was one of high tension throughout, it never became dicrotic (Fig. X, 1).

No reaction was obtained, however, with the guaiacum test. At one time he had some slight œdema of his lower extremities. As he became convalescent the albumen gradually diminished, and at last disappeared from his urine. Fig. X, 2, illustrates his pulse at this period; it is perfectly normal. He went away to the country during his convalescence, and while there, according to his own account, he had a relapse of his fever.

When he first came to report himself, two months after his discharge, his urine was free from albumen, but gave a slight reaction with the guaiacum test. His pulse (Fig. X, 3) shows a decided increase of tension. In a fortnight's time he returned again, not feeling so well; he thought he had

FIG. X.

Commencing cirrhosis?



1. During enteric fever. Albumen. No blue.

2. Convalescent. Urine normal.

3. Two months after. No albumen. Faint blue.

caught cold. He complained of weakness and general malaise; his urine now gave a distinct blue reaction with the guaiacum test, and a faint trace of albumen was also discovered. This case, I have but little doubt, was one of commencing cirrhosis of the kidney. He still continues under observation.

Finally, while unwilling to lose the association of the name of Bright, with kidney disease, I agree with many others in thinking the term is too general for scientific purposes, for as applied by some it has become synonymous with "albuminuria." I venture to suggest, therefore, that the terms Acute and Chronic Excretory Congestion concisely convey a more extended and accurate idea of the organs affected and of the pathological changes which occur in the two conditions discussed in this paper than many of those at present in use. The two other forms of kidney frequently included in the term Bright's disease, namely, the waxy and true fatty kidneys, are, of course, of an entirely different nature, and form only part of the general waxy and fatty degenerations which affect the tissues generally.

ON

SEPTIC DISEASE

IN AND OUT OF HOSPITALS.

BY

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(Received April 14th—Read May 12th, 1874.)

THE large death rate which follows operations in hospitals, and especially in large hospitals, has of late engrossed a great deal of attention. The subject was as it were reintroduced by Mr. Erichsen at the beginning of the year in a series of papers entitled "Hospitalism,"[1] and under another form and with a totally different intention by Mr. Prescott Hewett in his presidential address at a sister Society[2] with the title "Pyæmia in Private Practice."

It must, however, be in the memory of every Fellow of this Society that several years ago Sir J. Simpson published a series of papers on this subject, whereof Mr. Erichsen's are as it were a continuation, following as he does the same mode of reasoning, method of interpreting statistics, &c.

[1] 'British Medical Journal,' January and February, 1874.

[2] Address of the President of Clinical Society.

Hence if in the first few pages of this paper I refer only to Sir J. Simpson's views, it is with the idea that the same remarks will apply to the later writer.

Sir J. Simpson published his papers on "hospitalism" chiefly during the year 1869; they created much attention at the time, his numbers were greatly objected to and a good deal of controversy ensued. With characteristic energy he had amassed a large array of figures concerning the results of the four principal amputations; these he sorted into classes according as the operation was performed, in larger or smaller hospitals, or in private practice, and then adding up the number of each class he found, or thought he found, that in larger hospitals the death rate is very much higher than it is in smaller ones, and these higher than in the very smallest; while in isolated cottages, however cramped and ill-kept, the result is best of all. His deduction resulted in the coinage of the word hospitalism, whereby he intended to designate a certain injurious septic influence which is more intense in larger than in smaller institutions and which kills operation-patients in direct ratio with the number of beds in the hospital, by producing septicæmia, or pyæmia, erysipelas, gangrene, tetanus, and to a certain extent exhaustion.

Now, it appears to me that this is the very abuse of statistics. To take one only of a set of circumstances and to ascribe all the augmentation and diminution of death rate, all the favorable or unfavorable returns, to that one circumstance alone seems the very reverse of logical.

It is not my intention to produce other and I think more reliable statistics, as a mere quotation of figures against figures could lead to nothing, but I propose to accept Sir J. Simpson's returns regarding hospitals, and to treat them in a different manner. His three first orders contain four large London hospitals, to which subsequently five others are added. Secondly, four large provincial hospitals containing between 200 and 300 beds. Thirdly, eighteen provincial hospitals with from 100 to 200 beds. Simpson added together the returns from each class and

thus produced his result. But if we take each hospital separately we are driven to a very different conclusion (see Table I), as a glance at the table will show. It is compiled from Simpson's numbers, but the classes are divided according to the death rate, not according to the number of beds, showing each hospital separately.

TABLE I.

Number of beds.	Name of hospital.	Percentage of deaths after operation.
300	Middlesex	75
200	Westminster	53·3
120	Buxton	50
510	London	47·3
100	Royal Free	46·4
250	Newcastle Infirmary	42·5
134	North Liverpool Infirmary	40·2
170	King's College	40
350	St. George's	38·6
580	Guy's	38·2
650	St. Bartholomew's	36·6
223	Birmingham General	31·5
109	Greenock	31
140	Leeds	29
270	Liverpool General Infirmary	28·9
165	Sussex County	27
120	South Liverpool	26·2
120	Bath	25·4
125	North Staffordshire	24·5
150	Norfolk and Norwich	23·5
150	Bristol General	23·4
150	Derbyshire	19·4
140	Shrewsbury	19
150	Chester	18
165	St. Mary's	17·6
149	Radcliffe	16
242	Bristol Infirmary	15·7
142	Nottingham	14·9
108	Rochester	13·3
120	Reading	11·2
120	Kent and Canterbury	5

We find—

Three hospitals with 50 per cent. of deaths and over, containing 300, 200, and 120 beds.

Five hospitals with 40 per cent. of deaths and over, containing 510, 250, 170, 134, and 100 beds.

Five hospitals with over 30 per cent. of deaths containing 650, 580, 350, 223, and 109 beds.

Eight hospitals with over 20 per cent. of deaths containing 270, 165, 150 (twice), 125, 120 (twice).

Ten hospitals with under 20 per cent. of deaths containing 242, 165, 150 (twice), 149, 142, 140, 120 (twice), and 108 beds respectively.

Thus I make five groups, and in considering them it is evidently impossible to find any close connection between the size of the hospital and its mortality. Some of the first and second groups are formed by the smallest hospitals. The two largest hospitals in England are in the middle group. A large hospital with nearly 250 beds is in the lowest class. But we can go further. Omitting the Middlesex and St. Mary's Hospital, which evidently had runs of fortuitous good and evil times in the one and two years during which Sir James gives their statistics, we find that of the nine metropolitan hospitals quoted, the largest has the lowest death rate, while the smallest, with only 100 beds, is among the most lethal. Liverpool maintains three hospitals; the largest and the smallest, with 270 and 120 beds respectively, have an almost equal percentage, while one intermediate in size has a much higher mortality. In Bristol are the infirmary with 242 beds and the general hospital with 140. The large institution has 15·9, the smaller 23·4 per cent. of deaths.

Evidently Sir James Simpson was quite wrong in his idea that the mortality of amputations is regulated by the size of the hospitals in which they are performed.[1]

Nevertheless, it must be conceded that metropolitan hos-

1 In the table and subsequent remarks I have omitted the Scotch hospitals quoted by Sir J. Simpson, for reasons that will presently appear.

pitals (including the Scotch), and indeed, as a rule, institutions with more than 250 beds, have a large death rate; are we, therefore, to jump with Sir J. Simpson straight to the conclusion that the number of deaths is caused by the number of beds, or shall we, more philosophically, take other matters into account? For instance, every large hospital is, by the very force of circumstances, situated in a large town, and in large towns the death rate is higher than in rural districts, at the rate of about 5 per 1000. If this be the additional proclivity to death among the healthy outside, what additional tendency would there be in the wounded inside a hospital? yet this is less unimportant than the following considerations.

Every hospital surgeon must be aware that, quite independently of any injurious influence in his wards, his operations at certain times will not do well, and he postpones as much as possible any interference with the knife. This state of things corresponds with an unusual flux to the hospital of applicants with diffuse phlegmonous inflammations, erysipelas, perhaps even a case or two of pyæmia. Also he will find that any erysipelas admitted from without has a great tendency to spread; in fact the state of atmosphere outside, and, since the building derives its air from without, inside the hospital also, is such as to foster these morbid agencies. The condition, although it will at times be thus accentuated, is not a mere occasional, but is a constant accompaniment of residence in crowded houses and neighbourhoods.

If we examine into the returns of the Registrar-General, we find that the number of deaths from erysipelas increases with the number of persons residing on every square acre, not in absolute proportion, since many other circumstances interfere, but still in very close ratio. We may learn from these returns the varying percentage of deaths from this cause in towns and in country.

In London (the part which lies in Middlesex) the deaths are 18·5 per 100,000, in the rest of the county 10, but the districts here are rather thickly populated and hardly rural.

In Surrey, St. Saviour's, and St. Olave's, the death rate is 17; in the rest of the county 7·6.[1]

Towns.		Rural.	
Nine towns in Kent	15·7	Kent	8
Bristol, Gloucester, Clifton	18·8	Gloucestershire	7
Birmingham	14·5	Warwickshire	8
Three towns in Notts	14·4	Nottinghamshire	6

Per 100·000.

These are sufficient examples (though they might be indefinitely increased in number and minutiæ) to show the effect of aggregation, and it need scarcely be pointed out that the effect falls not on the occupiers of large houses in wide streets and squares, but on the dwellers in single rooms of small tenements situated in narrow lanes and courts. This influence I would call "urbism;" it produces, as the returns show, a great receptivity to septic forms of malady. It is impossible to go further than this; we none of us know what erysipelas is, but we see that overcrowding, with its attendant evils, produces a state of body which renders it prone to septic disease, and our town hospitals, especially our metropolitan hospitals, fill their wards with this overcrowded class. If, then, this portion of a town population, unwounded and unhurt, die of septic disease in twice or thrice the proportion in which countrymen die of it, by how much larger a ratio will this people, injured and wounded, succumb to such conditions? This is a simple problem in equations with which the number of beds in hospital has, as we have seen, nothing to do, but for the solution of which certain factors are absent, therefore only a proximate answer can be given, and this I will do by a rearrangement of Simpson's Tables.

The great obstetrician took most of his hospital returns from the year 1862 to 1868 inclusive, and I have compiled

[1] The numbers are for the year 1871.

Table II by extracting the death rates from erysipelas during those years for the districts in which the hospitals are situated, and constructing their average. The returns from the metropolitan hospitals are combined, omitting the Middlesex and St. Mary's, which are, as above stated, hardly reliable. Certain other hospitals are of necessity omitted, as the Scotch and some in subdistricts for which I have no annual reports. The table is arranged from above downwards in the order of the erysipelas death rate.

TABLE II.

District in which hospital is situated.	Deaths from erysipelas per 100·000.	Percentage of deaths in hospital after operation.
Metropolitan	17	42·8
Liverpool	14·8	26·2—28·9—40·2
Leeds	14·7	29
Stafford	13·5	24·5
Birmingham	12·4	31·5
Rochester (Medway)	11·2	11
Derby	10·5	19·4
Chester	9·5	18
Oxford	9·5	16
Newcastle	9	42·5
Reading	8·8	11·2
Canterbury	8·5	5
Norwich	8·3	23·4
Bristol	7·7	15·7
Shrewsbury	7·7	19
Bath	7·7	25·4
Brighton	6·6	27
Nottingham	6	14·9

At first glance it appears from this table that the proportions between the percentage of erysipelas deaths in the districts, and of operation deaths in the hospitals, are very irregular. A second look will show that the irregularity is almost entirely produced by five hospitals (in italics in the table), Birmingham, Newcastle, Bath, Brighton, and

Norwich; the first four of these are referred to by Dr. Bristowe and Mr. Holmes, in the Government Reports on Hospitals, as much infected by pyæmia; the fifth is now in such condition that it has become a question with the governors whether it may not be necessary to pull it down entirely or in part. The other hospitals which may be assumed to be normal have a death ratio, which in some is rather above, in others rather below, double the erysipelas death rate of the locality (two are much under this ratio, and are peculiar in manners not necessary to discuss here). The general outcome of this Table, therefore, is that the mortality of amputations in hospitals, which are well ventilated, drained, cleaned, and otherwise well managed, declines with the local death rate from erysipelas or other septic disease, not in any strict ratio, since other matters interfere, but in a much closer proportion than has been I think hitherto suspected.

I have not the means of following this same comparison into the minute hospitals quoted by Sir J. Simpson, nor is there any necessity for doing so, as it is evident the same mode of reasoning carried into subdistricts and mountain villages would only bring like results further developed. Moreover, many statements in "*Hospitalism*" might be controverted were my intention merely to criticise that work. But I have desired to use that book only to show that in some sense—in the sense in which the word was coined—"hospitalism" does not exist. Bad construction, poisoning of wards by the dead-house, bad ventilation, overcrowding, &c., do exist, but these are no more essential to hospitals than hanging an innocent man—an event which has sometimes occurred—is an inherent portion of criminal legislation.

On the other hand "urbism" is a condition placed beyond any possibility of doubt by the Registrar-General's Tables, and by the calculations to be made upon their basis. It has, I believe, a large influence on the result of operations, and the knowledge of its existence may, I venture to hope, conduce to lessen the death rates from septic disease in our town hospitals.

To this part of my subject I shall in future revert, but I must

first limit the denial which I have given to the existence of hospitalism so carefully as not to be misunderstood. That a certain influence pervades surgical wards in which several suppurating wounds are collected is evident from the fact that we may not ovariotomise in such locality. That occasionally a ward may be infected with erysipelas and pyæmia—that some hospitals are habitually in a septic condition—is only too evident. But that a hospital properly tended and kept must of necessity be in a state to destroy a large proportion of operated patients is, I am sure, a fallacy, and a very mischievous one; since it would tend to make the surgeon acquiesce in a mortality which he ought with all his strength to combat.

ON A

DISEASE OF THE SKIN

PRODUCED BY THE

ACARUS FOLLICULORUM,

ILLUSTRATED BY CASES OBSERVED IN THE DOG.

BY

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COMMUNICATED BY

W. FAIRLIE CLARKE, F.R.C.S.

Received April 14th—Read May 26th, 1874.

Since Gustav Simon discovered the *Acarus folliculorum* in 1841 (see 'Müller's Archiv') much has been written about the anatomy and even about the development of this curious creature by Simon, Professor Erasmus Wilson, and others, but it has been generally regarded up to the present time as a harmless denizen of the sebaceous glands and hair-follicles of the skin.

The only person who has really described a diseased condition of the skin due to its presence is Dr. Gruby, who was professor of anatomy and histology in Paris in 1846, and who published his earlier observations in the 'Comptes Rendus,' March, 1845, and his later and more important ones in the 'Monthly Journal of the Medical Sciences,' (England) in November, 1846. Before, however, I speak

of Gruby's researches in detail, or the evidence which I have to adduce in confirmation of them, it may be best to lay before you briefly the opinions of other writers on the subject which I have been able to collect up to the present time.

Simon, the discoverer of the *Acarus* ('Die Hautkrankheiten durch anatomische Untersuchung erläutert,' 2te Auflage, Berlin, 1851), speaks doubtfully on the question whether it can produce a disease of the skin, and says (S. 319): "It is possible that the presence of the *Acarus* in very large numbers might give rise to comedones and acne pustules, though this cannot be decided for certain." Simon also questions the accuracy of Gruby's observations. Wedl ('Grundzüge der pathologischen Histologie,' Wien, 1854), S. 807, says: "The *Acarus* being unprovided with spines or bristles and living in the fat of the sebaceous glands produces no irritation in the skin, and could only if present in large numbers produce an irritation of the gland itself and the neighbouring tissues."

Professor Erasmus Wilson in his elaborate memoir on the *Acarus* or *Entozoon folliculorum* in the 'Philosophical Transactions' for 1841, p. 805, although he goes most minutely into the descriptive anatomy of the animal, has no reference to any disease occasioned by it; and in later editions of his work on diseases of the skin, *e. g.* the edition of 1868, p. 876, he says: The Steatozoon (*acarus*) would seem to give rise to no uncomfortable effects by its presence unless perchance it should multiply to such an extent as to become a source of irritation to the follicle." Mr. Wilson states that he "had only examined healthy people when he wrote, and had not had recourse to a skin affected with acne," and he does not allude to the possibility of its producing a disease in animals. Förster in his 'Lehrbuch der pathologischen Anatomie' (8te Auflage, 1868), says (S. 548): "The *acarus* (*Haarsackmilbe*) is always found accidentally (ein zufälliger Befund), and appears not to give rise to any alterations in the skin."

Professor Hebra, of Vienna, in the last German edition

of his 'Lehrbuch der Hautkrankheiten,' 1872, S. 98, speaks as follows: "In my opinion the *Acarus folliculorum* can be regarded neither as the cause of comedones nor of an acne, because one is less often successful in extracting it from comedones or acne tubercles than from the follicles of persons with fine smooth skins, by scraping the skin with the back of a knife blade and examining the secretion which is thus squeezed out."

Lastly, Neumann, of Vienna ('Lehrbuch der Hautkrankheiten,' Dritte Auflage, 1873, S. 506), says decidedly: "The *acari* (*folliculorum*) have no further injurious influence on the skin (beyond occurring in it); according to Gruby they make the hairs fall out, which is a very improbable thing ("was sehr unwahrscheinlich ist.") Other English works on skin diseases beside Wilson's, as far as I know, merely allude to the *acarus, en passant,* and neither Hardy nor Bazin in France mention it at all, while Devergie merely quotes a passage from Gruby without expressing any independent opinion as to his facts. The larger number of authorities, therefore, are inclined to the belief that the *acarus* does not produce any skin disease at all either in man or animals, and the most recent writers, *e.g.* Hebra and Neumann, are the most positive of all of this, although apparently acquainted with the direct statements of Gruby, which have somehow failed to receive much credence either from his contemporaries or his successors. His writings have indeed been so nearly forgotten that it was with difficulty I succeeded in finding the reference to his original papers.

Dr. Gruby inoculated a "middle-sized" dog with the acari from man, and kept it under observation for two years. At the end of this time he states that "the animalcules have so increased as to occupy every follicle in his skin; he has lost all his hair, and the skin is as naked as that of an infant. The epidermis has been raised in various places, leaving the true skin exposed, and here and there considerable inflammation has been excited, causing scales of inspissated pus to form on the surface, and his general strength is now so reduced that he can scarcely

move." In this animal sections of the skin showed under the microscope enormous dilatation of the sebaceous follicle, with destruction of their cells, and with inflammation and small abscesses in the substance of the cutis. The hair-follicles were dilated, and there was loss of hair from atrophy of the papillæ, probably from the pressure of the acari which penetrated to their roots. "Thus," says Gruby, "a parasite which exists in man as a physiological condition occasions in the dog a disease of a very grave character."

In the course of the year 1873 three dogs were admitted at different times into the Brown Institution, Nine Elms, suffering from a disease of the skin, which consisted of loss of hair, scaliness, and pustulation somewhat resembling acne, and covering the greater part of the body, especially in the second of the three cases. All three died in a few weeks, in spite of all treatment, in a state of wretchedness and emaciation, apparently accelerated by their inability to keep themselves warm from loss of hair. In the first and third cases a careful post-mortem examination, made by my friend Mr. Duguid, the resident veterinary surgeon, to whose kindness I am indebted for the materials of this paper, showed no disease of any internal organ which would account for death. In the second there was inflammation of a few inches of the small intestine, whose origin was not very clear, but was apparently quite unconnected with the skin affection. The third dog died of gradual emaciation while under treatment with sulphur ointment and apparently improving. This dog, a piece of whose skin is before you, was in parts quite hairless, and covered on the back with crusts and scales of pus and exudation from the eczema caused by the irritation of the acari and the dog's scratching. He shivered continually though it was summer time, and seemed quite unable to keep himself warm.

Other dogs which were kept in the kennel with the second dog caught the disease from him, and had to be killed eventually. In the pustules with which this dog was studded acari were found in different stages of development, as well as ova, and full-grown acari with six and eight legs

respectively. The perfect animals in every respect resemble those of man. Pieces of skin were taken from each animal soon after death, and after hardening were examined microscopically.

All the three cases resembled one another in the enormous number of acari present, dilating the hair-follicles and filling the sebaceous glands, so as completely to destroy their secreting cells. The acari were found at all depths, both in the hair-follicles and the sebaceous glands. In all three cases inflammatory changes, of greater or less extent, were found in the cutis, arising from the irritation of the acari and the scratching of the dogs. These consisted of a small-celled growth around the blood-vessels in the papillæ and the upper part of the cutis, and of nodules of a new formation in the neighbourhood of the affected hair-follicles and sebaceous glands, whose size bore some relation to the extent to which the latter were diseased. There was also more pigment than usual in the lower layer of cells of the rete mucosum. The hairs were generally entirely lost, their papillæ atrophied, and even the root sheaths of their follicles in many instances reduced to a sort of thin fibrous layer. These changes will be more clearly comprehended by reference to the drawings of the microscopical preparations which accompany the paper (see Plate VI). In the first case alone nodules were found around the coils of the sweat-glands, deep in the corium, where no acari had penetrated; in fact, these animals appear scarcely ever to enter the sweat-ducts, for I have only seen one instance where such an entrance had apparently occurred. Probably their corkscrew mode of ending on the skin would prevent the acari from penetrating into them. I am inclined, therefore, to attribute this deep-seated inflammation to the irritation set up by the retained secretions of the sweat-ducts from pressure near the surface of the skin. The nodules around the sweat-glands consist (as represented in Plate VI, fig. 4) of a reticulum of nucleated cells as a matrix, with spherical lymph-corpuscles in their meshes (the so-called adenoid tissue), and resemble the similar nodules which occur in

other parts of the skin in chronic inflammations. Nodules of the same nature occurred just beneath the epidermis in the third case, and in their most advanced stage were found breaking down in their centre so as to form small abscesses, in which numerous acari were present.

With the evidence before us there can therefore be no longer any doubt that the *Acarus folliculorum* when it occurs in the dog is not by any means a harmless parasite, but, on the contrary, as dangerous to his existence as the *Acarus scabiei* is to the rabbit, and perhaps in rare cases to very young infants.

From the analogy of the disease in the dog, I cannot help thinking that, after all, some cases of acne in man may be due to the irritation of the acarus. I merely allude to the subject here, and hope to be able to offer some remarks on the subject at a future time.

Mr. Duguid informs me that, as far as he knows, the disease above described has no place in any work on veterinary medicine, and I believe that it has not been described as an independent disease before, for Gruby produced it by inoculation.

In conclusion, I should state that I was led to pay attention to the subject from finding that so many authorities doubted whether the *Acarus folliculorum* could produce a disease of the skin, and it was not until two of the cases had been examined and preparations made from their skin that I became acquainted with Dr. Gruby's writings, which confirm my own researches and receive confirmation in return from them.

The inquiry has been conducted in the laboratory of the Brown Institution, and I have to offer my best thanks to the authorities of that Institution not only for the opportunities of examining the animals which form the subject of the paper, but for much kind assistance and advice.

Postscript.—Since this paper was read, Prof. Simonds, of the Veterinary College, has called my attention to a paper of his in the 'Royal Agricultural Society's Journal' for

1865, vol. i. In it he describes a disease in dogs due to the *Acarus folliculorum*, and gives some directions as to treatment. Prof. Simonds has therefore anticipated a great deal that I have said in my paper, and to him is due the merit of first describing an idiopathic disease arising from this parasite. It is scarcely necessary for me to state that until I received Prof. Simonds' letter I had never met with any reference to his paper, nor had the slightest knowledge of its existence. Others beside myself seem to have similarly overlooked it, probably owing to its publication in a lay journal.—*June 14th*, 1874.

DESCRIPTION OF PLATE VI.

ACARUS FOLLICULORUM.

FIG. 1.—Section of skin to show loss of hair and dilatation of the hair-follicles, and sebaceous glands which contain acari, and in several places have quite lost their secreting cells. Some nuclear growth in the cutis, and pigmentation around the hair sheaths. (somewhat oblique section). Hartnack eyepiece III, objective 3.

FIG. 2.—Two sections from parts close to one another. 1. An immensely dilated hair-follicle whose structure is completely destroyed, and which is filled with acari and their *débris*. Two sebaceous glands open into its upper third, and though not much dilated contain acari. 2. A sebaceous gland dilated, and only filled with the *débris* of acari. By its side a hair passes up from a follicle which is still intact. Numerous nuclei in surrounding tissues. Hartnack eyepiece III, objective 4.

FIG. 3.—A much dilated hair-follicle with atrophied sheath and papilla. To show the acari highly magnified. Hartnack III, objective 7.

FIG. 4.—Several sweat-ducts cut transversely and obliquely (part of the coils of a gland). A nodule of inflammatory tissue (lymphatic reticulum, see p. 243) is seen filling a large part of the field. At its edges the reticulum, with lymph-corpuscles, is clearly seen, but towards the centre it becomes indistinct, and only the cells can be made out. In a further stage it would constitute a microscopic abscess. A blood-vessel (vein) is seen in section, and a capillary in its length in the nodule. The single layer of epithelium in the sweat-ducts, and the muscular fibres in their walls, are worthy of notice, as a similar arrangement occurs in many of the sweat-ducts and glands of man. This fact had been observed by Sharpey, Klein, and others, some time ago, but Prof. W. Krause ('Centralblatt für die Med. Wissenschaften,' 1873, No. 52, s. 818) describes it as a new discovery. Hartnack III, objective 7.

Fig. 1.

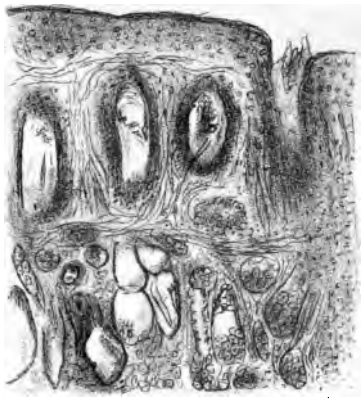


Fig. 2.

1.

2.

Fig. 3.

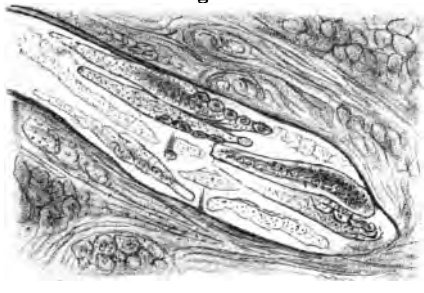
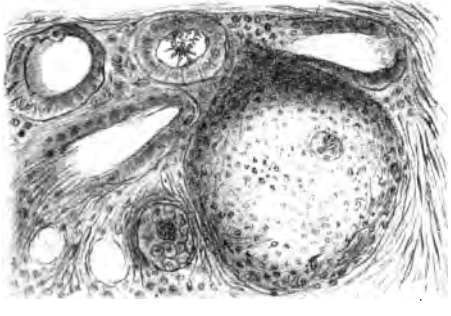


Fig. 4.



ON A CASE

OF

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS.

BY

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(Received May 12th—Read May 26th, 1874.)

For an opportunity of seeing the case during life, and of making an examination after death, we are indebted to Mr. Wm. Adams, under whose care the patient was. Mr. Adams has also furnished an account of the early history of the case.

Master W. E—, aged at the time of his death $14\frac{1}{2}$ years. Both parents are living and in good health. The father, an officer in the army, is a strong healthy man; the mother is a highly nervous hysterical woman. The only other child is a girl, a few years older, and in good health. No history could be obtained of any similar affection in collateral branches. In infancy the boy was well nourished and apparently healthy, but began to walk later than other children—not until he was two and a half or three years old, and then it was in a peculiar way.

About that time his calves were observed by his mother to be unusually large. As he grew older the peculiarity in his walk became more marked: he carried his body bent slightly forwards and walked with a swinging movement. He was never able to walk far, but when five years old could walk in the manner described about a mile. At this age the calves were noticed to be very large, the thighs were thought to be about the normal size. The arms were fleshy, but were not noticed to be particularly large.

When between five and six years old he became liable to fall in walking, and had great difficulty in getting up again.

A photograph, taken when he was between seven and eight years of age, represents him as a tall, well-nourished, and apparently strongly-built boy, but he was obliged to lean against a pillar for steadiness, and always used a stick in walking on account of his liability to fall. About this time the heels were observed to be somewhat drawn up. As this increased he became unable to walk, and after giving up walking he had some difficulty in straightening the knee-joints, and the elevation of the heels increased.

When nearly nine, in July, 1867, Sir William Fergusson was consulted, and advised division of the Achilles tendons, observing that if the boy could put the heels to the ground he would probably be able to walk. No operation was, however, performed, on account of domestic difficulties.

At the age of $10\frac{1}{2}$, in January, 1869, he was brought up to London to see Mr. Adams, who, thinking that division of the Achilles tendons would enable the boy to stand and walk, procured him admission into the Royal Orthopædic Hospital. At that time both calves were large and hard, the gastrocnemii being prominent and well defined in outline. The Achilles tendons were also tense and prominent, and the os calcis in each foot elevated so as to produce marked talipes equinus. The hamstring tendons were also tense so as to prevent complete extension of the knee. The thighs were small and the recti feeble.

He could, however, sit upright very well on a chair, and from this evidence of the state of the spinal muscles it was thought that he would be able to stand and walk upright if the heels could be brought down. The upper extremities were somewhat feeble. The biceps was noted as being particularly small and feeble, offering but slight resistance to passive extension. The pronators and supinators of the forearms were large, hard, and well defined. He could grasp with considerable power. The deltoid on each side was large, prominent, hard, and well defined in outline, and the arm could be held out in an extended position with considerable power. The pectoralis major was decidedly feeble and somewhat atrophied. The trapezius muscle was also somewhat feeble and atrophied, and there was some preternatural mobility about the scapula on each side, probably due in part to a feeble condition of the serratus magnus and latissimus dorsi muscles. The difficulty which the boy had in raising himself from the horizontal to the sitting posture was very striking, and appeared to depend, in part at least, on the weakness of the muscles about the shoulder-joint. When accomplished he could very well maintain himself erect in the sitting posture, and no spinal curvature existed.

During his stay in the hospital, from January to July, 1869, both Achilles tendons were divided by Mr. Adams. The hamstring muscles were subjected to forced extension, by which the knee-joints were straightened. The effect was that, with steel supports to the legs, carried up to the hips, he was able to walk about the ward with assistance. He was unable to use crutches on account apparently of the weakness of the muscles about the shoulder-joints; and with this amount of improvement he returned to the country in July, 1869. Just before leaving town he was seen by Dr. Radcliffe.

Soon after his return home the mechanical supports were unfortunately discontinued, and the boy never stood or walked again. From this time there was a steady loss of

power with wasting of muscles both in the upper and lower extremities. The contraction of the knee-joints returned and increased. In 1871 he was quite unable to straighten the knees, and could move only his toes and ankle-joints. The wasting and weakness of the arms increased until he ceased to be able to raise them even to feed himself, and could only put food into his mouth by resting his hand upon his raised and bent knees and lowering his head. The right arm continued stronger than the left.

In the summer of 1872, being then nearly 14 years of age, he had an attack of gastric fever, which weakened him considerably. Subsequently he suffered from bronchitis, which never completely left him. He was then attended by Drs. Magrath and Workman, of Teignmouth. The general wasting and debility increased, and some difficulty in swallowing came on and continued.

In January, 1873, he was again brought to London to see Mr. Adams, at whose request he was seen by Dr. Radcliffe and by Dr. Richardson. His condition was then noted to be as follows:

Mind quite unaffected. A cheerful clever boy; fond of reading. Voice high-pitched. No apparent weakness of any muscle supplied by cranial nerves. Special senses unimpaired, optic discs normal. No wasting of face; no want of expression in it. Tongue protruded straight and moved freely, unwasted. Complaint was made of slight difficulty in swallowing. The palate appeared to move freely.

Arms.—No power of moving shoulder- or elbow-joints. The wrist-joints could be moved with tolerable freedom, and the fingers well, so that he could write and draw. The elbows were flexed at nearly a right angle, and could not be straightened. All muscles of the arms were greatly wasted, deltoid, biceps, triceps, and forearm muscles. The wasting, though great, was not extreme. The muscles of the left arm were rather less wasted than those of the right. No fibrillation was seen.

Trunk and back muscles all considerably wasted, though not quite as much so as those of arms.

Spine presented a moderate degree of lateral curvature, the convexity being to the right.

Legs.—No power over hip- or knee-joints. The ankles could be moved a little, but not much, the calf muscles having again become contracted, and having reproduced the state of talipes equinus which had been removed by tenotomy four years previously. The toes could be moved fairly well. The hip-joints were flexed, the thighs being brought up towards the abdomen, and the knee-joints bent at a right angle: both were rigid from muscular contraction and the rigidity could not be overcome.

All the muscles, both of thighs and legs, appeared much wasted; there was scarcely any prominence of the calves.

Sensibility was everywhere unimpaired. The sphincters were unaffected.

From this date no marked change occurred in his muscular symptoms. His cough became more troublesome and evidence of consolidation in the lungs was found, and his strength rapidly diminished. Towards the end he complained of some increase in the difficulty in swallowing. He died on March 19th, 1873, aged 14½ years.

The post-mortem examination, forty hours after death, was made under circumstances of some difficulty.

The wasting and position of the limbs were the same as noted during life.

All the thoracic and abdominal viscera were healthy with the exception of the lungs. Throughout both lungs were thickly scattered areas of lobular pneumonia, grey in colour, many of them breaking down. Some larger areas of consolidation in the right lung emitted an extremely offensive gangrenous odour. No grey granulations could be seen.

Muscles.—Portions of muscle were removed from the gastrocnemius, the deltoid, and the biceps, on each side.

The gastrocnemius muscles were small, not more than one third of the normal size. Each muscle presented the appearance of a mass of adipose tissue. On section

it was pale yellow in colour and greasy to the touch. No trace of red tint could be seen in it. Under the microscope it consisted for the most part of distended fat-cells, which lay between and separated widely bundles of muscular fibres. The fat-cells were quite similar to those constituting an ordinary lipoma; they varied in size from $\frac{1}{250}$ to the $\frac{1}{1000}$ of an inch. In a few a nucleus could be seen. They contained, when fresh, no crystals. The amount of connective tissue between them was small.

The muscular fibres ran, several together, through the adipose tissue. Some were of normal size, others much less. Their diameters varied from the $\frac{1}{1000}$ to $\frac{1}{3500}$ of an inch. In many places the narrower fibres lay beside the wider ones. Many fibres presented variations in width, being narrower at one place than at another. In some this variation in width was sudden, as at Plate IX, fig. 7. The transverse striation was for the most part, in the narrower as well as in the larger fibres, perfectly distinct. The striæ were, in the larger fibres, about $\frac{1}{1200}$ inch distant from one another, but in some of the smaller they were farther apart. Here and there, in the narrower fibres, the striæ had disappeared, being replaced by granules distributed uniformly through the fibre. In a few of the larger fibres the striæ were assuming a granular aspect. In a few, both large and small, the striæ seemed to be disappearing, fading away, without becoming granular. In no case were any fat-globules seen within the fibres. No distinct division of fibres could be detected. Accompanying the bundles of muscular fibres, changed and unchanged, was a good deal of connective tissue, the fibres of which lay parallel to the muscular fibres. Some of this had the aspect of ordinary fibrous tissue, but much of it contained small oat-shaped nuclei, and had very much the appearance of empty sheaths of muscular fibres (Plate VII, fig 1.) Indeed in some places it was evidently the case, for on following such fibres for a little distance transverse striation gradually showed itself, at first faint, then more distinct, until the aspect of a distinct fibre was assumed.

Deltoid.—The deltoid, much below the normal size, had a reddish tint, though considerably paler than natural. The microscopic appearances were similar to those of the gastrocnemius, but less advanced. In places the fibres were separated by a large amount of fat; in other places there was little fat, but much fibrous tissue between them. They presented even greater variation in size than those of the gastrocnemius, being from the $\frac{1}{500}$ to the $\frac{1}{4000}$ inch in diameter. The average size, however, was about $\frac{1}{1000}$ inch. The striæ were farther apart in the narrower than in the larger fibres, the variation being between the $\frac{1}{14000}$ and $\frac{1}{7000}$ inch distance.

The *biceps* on each side presented naked-eye and microscopic appearances very similar to those of the deltoid, but the amount of fat was less and no fibres were seen more than $\frac{1}{1000}$ inch in diameter.

Tendons.—Both Achilles tendons were natural on external appearance, in outline, and thickness. When divided longitudinally it was evident that $1\frac{1}{2}$ inch of new tendon had been formed and inserted between the ends of the old tendon, where they had been divided four years previously, the new portion being readily distinguishable from the old by the grey colour and translucency of its tissue as compared with the opaque pearly lustre of the old tendon, and by the absence of the appearance of longitudinal striation which characterised the old. The line of junction, both above and below, was still distinctly traceable, the junction being effected by a process of dovetailing, the translucent tissue of the new tendon being inserted betw the separated fibres of the old tendon. The naked appearances in this case were precisely similar to those scribed by Mr. Adams in his work on the 'Repara Process in Human Tendons.'

Nervous system.—Nothing unusual was found in eitl the external or internal appearance of th ·····in and medu oblongata.

The spinal cord, as it was remo seemed perfectly healthy and of nor

after being very carefully hardened in a solution of chromic acid, thin sections made in every region revealed, under the microscope, varied and extensive lesions.[1] These lesions began, from above downwards, at the level of the second cervical nerves. There was disintegration, in a greater or less degree, of the lateral grey network which is so conspicuous in this region between the caput cornu posterioris and the *tractus intermedio-lateralis*, and through which the spinal accessory nerve makes its way into that tract. The tract itself was to a certain extent in a state of incipient disintegration, and on one side was, in many sections, traversed by a dilated and congested blood-vessel around which the tissue had begun to suffer. The white columns were not perceptibly altered, but one lateral half of the anterior white commissure was entirely destroyed and replaced by granular *débris*, by exudations and extravasated blood-globules which also filled the triangular space at the bottom of the anterior median fissure.

On descending through the rest of the cervical region the same kind of morbid changes, to a greater or less extent, were found at different levels, while the anterior and central grey substance on each side was the seat of extensive disintegrations. A similar lesion existed in some of the posterior nerve-roots near their entrance into the caput cornu posterioris, as may be seen in Plate VII, fig 4, at *n*, where the fibres are completely broken up and thus severed from the gelatinous substance, *o*, of the posterior cornu.

Both the lateral and posterior white columns were in many sections much damaged by sclerosis, consisting of a variable degree of disintegration of the myelin or white substance of the longitudinal fibres. The lesions of the grey substance are seen in fig. 1, which represents

[1] In order to avoid the error (which is not possible with those observers whose experience in the healthy and morbid anatomy of the cord is great) of mistaking breaches of tissue produced in making sections, for morbid changes, it has been the practice to examine with a powerful lens the cleanly-cut surface of the cord where the section has been made.

a transverse section at the upper roots of the sixth cervical nerves. At *a* is a blood-vessel surrounded by a light area containing granular exudation and partially disintegrated tissue. Similar spaces are seen at a^2, a^3, a^4, on the left side, and on the right side several areas of incipient disintegration may be seen in the cervix cornu, a^1. But the most striking lesion was in the anterior commissure, *b*, the left half of which was entirely destroyed. Its place on that side was occupied by granular exudation and by extravasated blood-globules, *c*. A little lower down in the cervical enlargement the destruction of these parts was still more extensive. Plate VII, fig. 2, represents the anterior and posterior commissures with the inner portion of each lateral half of the grey substance more highly magnified than fig. 1. The anterior commissure, *b*, extends very little beyond the median line, on the left side, the rest being completely destroyed. In front of it is a mass of granular exudation, *d*, and a blood-vessel, *e*, entering at the anterior median fissure, *f*, extends into a large area of disintegration, *g*, at the inner portion of the lateral grey substance, the vessel itself sharing in the destruction. At *h h*, just where the anterior commissure winds round the inner side of the anterior cornu, there are two considerable tracts of disintegrated tissue. In some sections of the cervical enlargement the *posterior* commissure was also a little damaged, while in others both commissures were uninjured. In the triangular space at the bottom of the anterior median fissure a considerable extravasation of blood was very common. In the base of the posterior cornu on the right side, at the point *p*, in Plate VII, fig. 1, there was a remarkable area of disintegration surrounding a tubular cavity (see Plate VII, fig. 3). At the outer border of this area, *q q q q*, the grey substance was easily seen to be in process of disintegration, and farther in, this process had gone on to the formation of a pellucid and coagulable fluid surrounding a cylindrical tube, *r*, which was lined by an extremely thin layer of granules.

In the upper portion of the dorsal region the morbid

changes were less frequent and extensive, but here and there the anterior white commissure was partially destroyed. In the middle and lower portions of this region, however, the lesions were greater than in any other place. The lateral grey substance on the outer sides of the posterior vesicular columns, including the *tractus intermedio-lateralis*, was severely damaged by disintegration, while the anterior white commissure was in many sections wholly or partially destroyed. At the lowest part of this region and the commencement of the lumbar enlargement the lesions were most extensive and striking. Plate VII, fig. 5, represents a transverse section of the grey substance in this locality. The central and lateral parts on each side, *i i*, were severely damaged by softening and disintegration, presenting the appearance of a piece of cloth irregularly worn into holes. To the naked eye there appeared to be a cavity on each side occupying the central part of the grey substance.

These large areas of lesion, however, were still traversed by many fibres whose course is almost entirely peculiar to this particular portion of the cord. One set of these fibres is derived from the posterior roots of the nerves, as seen at *j*, and curves forwards and inwards to a point, *k*, in front of the posterior vesicular column, *l*. Other sets of fibres proceed from the anterior part of the lateral column and from the *tractus intermedio-lateralis*, and also from the posterior transverse commissure, and run transversely inwards and outwards to the same point, *k*, where both sets become longitudinal in their course along the grey substance.[1] As in the cervical region, the anterior commissure was often greatly damaged, and in some sections, as shown in Plate VIII, fig. 6, only the central portion, *b*, remained, its lateral portions, with some of the contiguous grey substance, having been almost entirely destroyed.

[1] The course of these fibres, the functions of which must be of great importance, has not been hitherto described by any anatomist. An account of them, and of their relations to surrounding parts, will shortly be communicated to the Royal Society.—J. L. C.

In the middle and lower parts of the lumbar enlargement the lesions were less serious. The grey substance was only slightly affected here and there by small patches of incipient disintegration, and the anterior commissure was much less damaged than in the other regions of the cord; but some of the nerve-cells were evidently wasted to a certain degree; their processes were shrunk, and their angles were more obtuse than natural. The anterior roots of the nerves were in many places injured by streaks of disintegration of variable size, and in some sections the fibres at their origin, as well as the border of the anterior horn from which they sprang, were completely destroyed, as shown at *m m*, Plate VII, fig 5, where the myelin or white substance of the fibres is stripped from their axes-cylinders and appears in the form of globules.

In the lower portion of the lumbar enlargement and in the conus medullaris the lesions of the grey substance were severer and more extensive. The central part of the anterior cornu and the outer part of the cervix cornu posterioris were very much damaged by continuous disintegration. In some of the sections at the level of the upper sacral nerves scarcely any large nerve-cells could be seen in the anterior cornu. Some of the nerve-cells were likewise atrophied, and contained more pigment than natural.[1]

Of the identity of the disease in this case with that described by Duchenne under the name of "pseudo-hypertrophic paralysis," there can, we think, be no question, although at the time of death the muscles were all smaller and not larger than the normal. At an earlier period the muscles of the calf had presented a decided increase in bulk, and this, together with the naked-eye and micro-

[1] It may be well to state that a piece of spinal cord about an inch long was taken from a patient of Mr. Adams about three years back, and sent to me for examination, but it had been so rapidly hardened in strong solution of chromic acid that it was impossible to make sections on account of its friability. In partial sections, however, I discovered unmistakable areas of transparent granular disintegration. The case was a typical example of pseudo-hypertrophic muscular paralysis.—J. L. C.

scopic appearance of their substance, the enormous accumulation of fat between the bundles of muscular fibres, sufficiently characterises the case as an instance of that disease.

It further agrees with most of the recorded cases in sex (four out of five being males), in the age at which the symptoms commenced, and in the early and chief affection of the gastrocnemii and solei muscles.

A condition of atrophy in other than the calf muscles is not unfrequent in this affection. Out of 81 cases collected by Friedreich some muscles were atrophied in 37; most frequently the pectoral muscles, next in frequency the muscles of the shoulder, back, and upper arm; least frequently those of the thigh, forearm, and hand. We believe that in no case hitherto recorded has there been atrophy of the muscles of the calves, previously increased in size.

In several of the cases on record, especially, for instance, in that described by Cohnheim, the identity of structural change in the muscles enlarged and in those atrophied was clear, and its characters corresponded in almost every particular to those presented by this case.

In the two post-mortem examinations made some years ago by Dr. Meryon, and in the more recent examinations by Cohnheim and Charcot, no structural change in the spinal cord was discovered. Mr. Kesteven has described, in a case of imbecility associated with this disease, dilatation of the perivascular canals and spots of granular degeneration, but none of the latter exceeded in size $\frac{1}{50}$th of an inch, and they are described as being in the cord "few and far between." Certain pathological appearances were found by Barth[1] in the cord of a man who died, aged 44, having presented for three years the symptoms of this disease, enlargement of some and atrophy of other muscles, the microscopic structure of both being characteristic of this affection. The changes consisted of "partial degeneration of the antero-lateral columns, due to proliferation of the neuroglia and the replacement of nerve-tubes by a

[1] 'Archiv der Heilkunde,' xii, 1871, p. 126.

finely granular and very vascular substance containing corpora amylacea. The ganglion-cells of the anterior horns were diminished in number throughout the cord. Corpora amylacea also lay between the fibres of the anterior roots of the nerves, the posterior roots being normal. Both anterior and posterior roots were surrounded by much adipose tissue, which filled also the intervertebral foramina and the interior of the vertebral canal."

From this case that which we have recorded above differed in the absence of any increase in the adipose tissue outside the cord and in the chief and considerable affection of the grey matter.

DESCRIPTION OF PLATES VII, VIII, & IX.

Microscopical Appearances of Spinal Cord and Muscles in a Case of Pseudo-hypertrophic Muscular Paralysis.

Plate VII.

Fig. 1.—Transverse section of grey substance of spinal cord at lower part of fifth cervical nerves. × 10.

Fig. 2.—Commissural portion, a few sections lower down. × 25.

Fig. 3.—Area of disintegration, inner part of lateral grey substance, lower cervical region. × 35.

Fig. 4.—Extremity of posterior cornu, with the corresponding posterior root interrupted by disintegration. × 200.

Fig. 5.—Anterior root, lumbar region, interrupted by disintegration.

Plate VIII.

Fig. 6.—Transverse section of grey substance at the highest part of the lumbar enlargement.

Plate IX.

Fig. 7.—Portion of gastrocnemius muscle, showing the narrowed muscular fibres, connective tissue, and nuclei between the adipose tissue. × 200.

Fig. 8.—From the same muscle, healthy-looking fibres, accompanied by increased quantity of connective tissue.

Explanation of Letters.

a^1, a^2, a^3, a^4. Areas of disintegration in the grey substance of the spinal cord. Fig. 1.

b. Anterior commissure.

c. Extravasated blood-globules.

d. Granular exudation. Fig. 2.

e. Blood-vessel.

f. Anterior fissure.

g. Large area of disintegration.

h. Do., less advanced.

i. Lateral portions of grey substance. Fig. 6.

j. Some of posterior roots of nerves.

k. Point where thin fibres become longitudinal.

l. Posterior vesicular column.

m, m^1. Extensive disintegration at origin of roots of anterior nerves. Fig. 5.

n. Disintegration of a large posterior at its entrance into the point of posterior horn.

o. Point of posterior horn.

q. Remarkable area of disintegration surrounding a tubular cavity, *r*, at the base of posterior horn. Fig. 3.

Fig. 1.

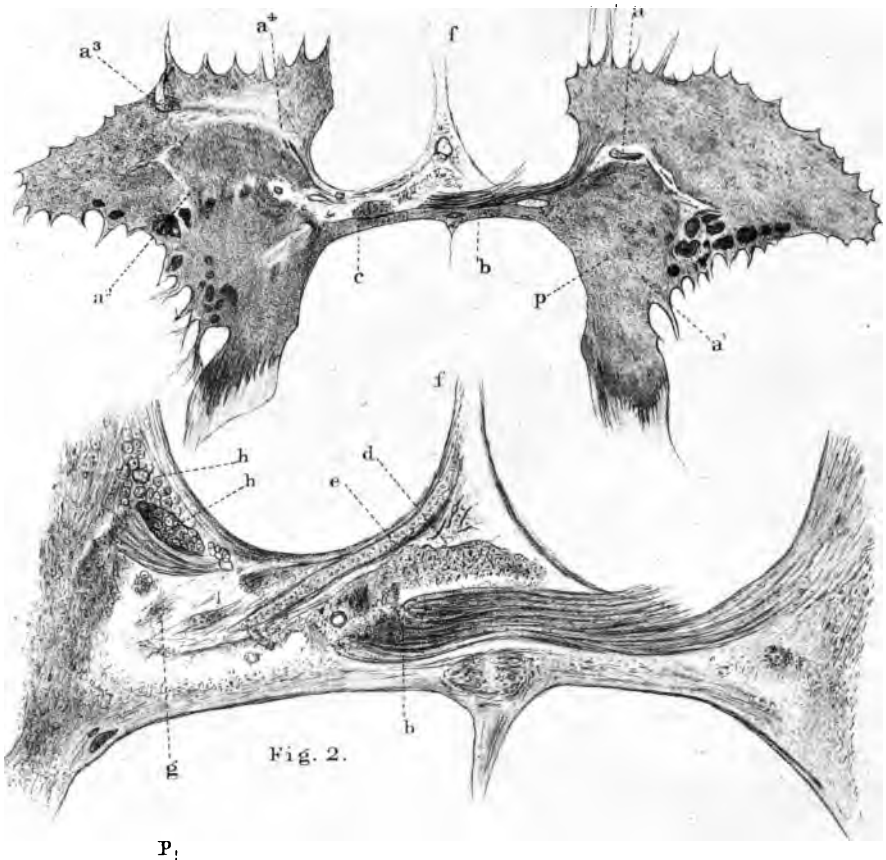

Fig. 2.

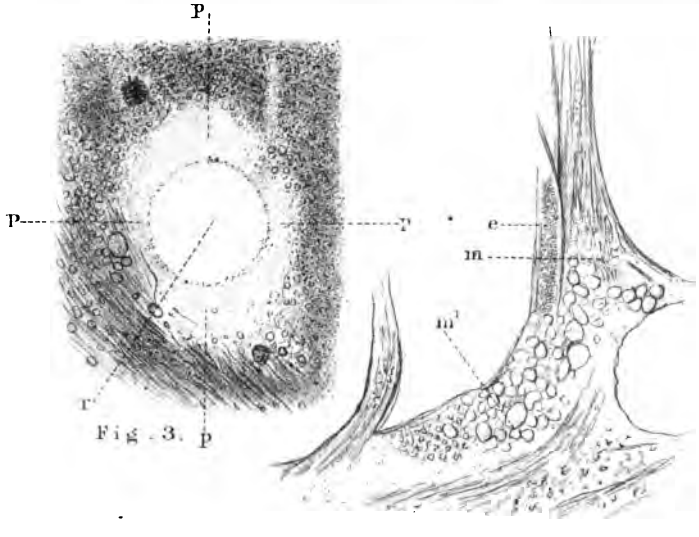

Fig. 3.

Fig. 5.

Fig. 4.

W.R.Cowers del. G.H.Ford lith.

Mintern Bros. imp.

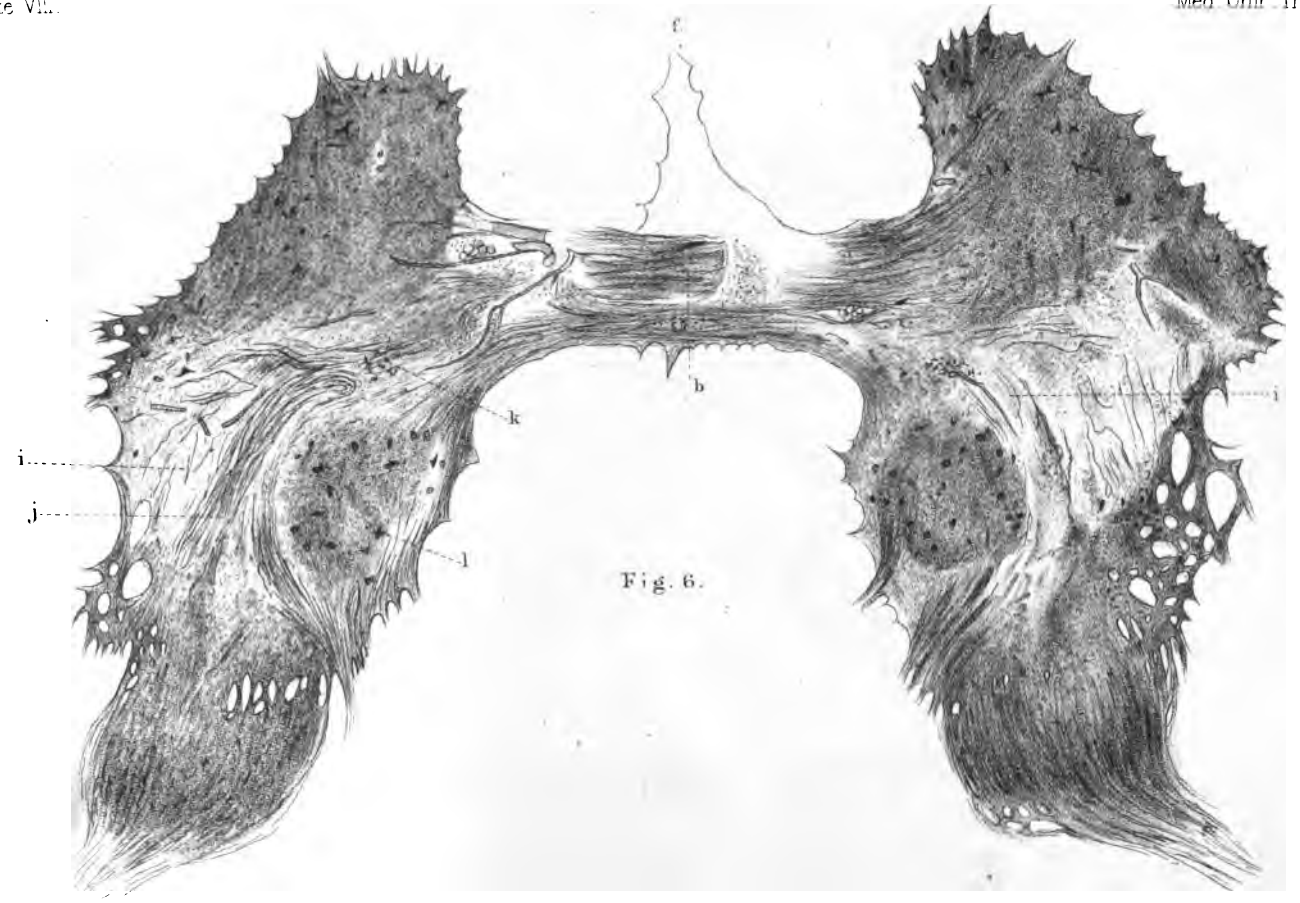

Fig. 6.

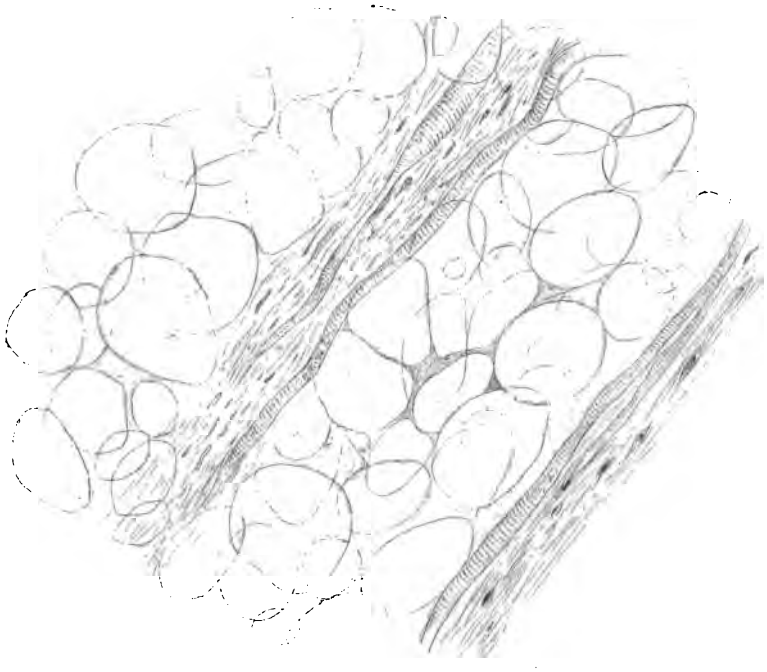

Fig. 7.

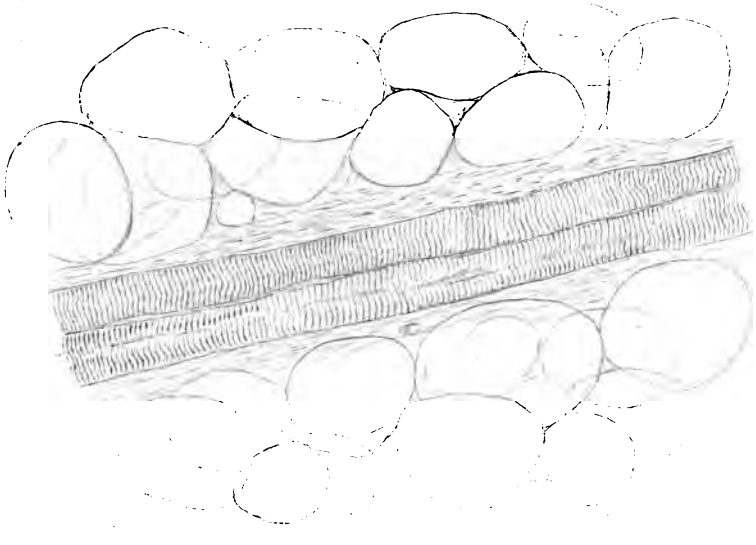

Fig. 8

A CONTRIBUTION

TO THE

NATURAL HISTORY OF PULMONARY CONSUMPTION,

CONSISTING OF AN ANALYSIS OF

ONE HUNDRED MALE CASES OF HÆMOPTYSIS.

BY

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(Received March 9th—Read April 28th, 1874.)

THE primary object of this investigation has been to assist, by clinical observation, in settling the important question of the true position of hæmoptysis in the natural history of pulmonary consumption.

With this intention, the cases discussed in this paper were recorded upon a plan of inquiry so designed that it should, as far as possible, avoid the chance of prejudice from preconceived opinions and impressions, and include all the facts essential to the investigation in *a tabular form*.

With so ample a field for observation as is afforded by the Royal Hospital for Diseases of the Chest, it would have been easy to accumulate a much larger number of cases, if I could have entrusted the work to an assistant. But feeling

that the whole value of the investigation depended upon my being able to guarantee that the histories of the cases had been sifted and verified in the most rigorous manner, there was no alternative but to do all the work myself.

I regret that this necessity and the small amount of leisure at my command have obliged me to limit my record to 100 cases of hæmoptysis—a number too small to form more than "a contribution" to the statistics of the subject. I trust, however, that if the plan which I have adopted should be thought worthy of imitation, other observers, with more time at their disposal, will follow it up, and thus accumulate a sufficiently large mass of comparable facts, arranged on a uniform plan, to form a safe basis for final conclusions.

Having decided upon the plan for the tabulated records, it was next of the highest importance that the cases should be representative of the ordinary average varieties of hæmoptysis, absolutely free from any selection dictated by special views, and yet, that they should be selected, in the sense of each containing the elements essential to the leading points of the inquiry. To obtain these ends the following precautions were rigorously carried out.

All cases occuring in my practice at the Royal Hospital were asked if they had ever expectorated any blood. If the answer was "Yes," the case was sifted in the following manner:

1. All cases were rejected in which the blood had never been seen in any other form than *streaks in the phlegm*.

2. All cases were rejected unless the heaviest weight before the occurrence of hæmoptysis could be stated from *actual weighing*, and reasonable evidence given as to whether this had been the average weight up to the time of first hæmoptysis.

3. All cases were rejected who could not stand a searching cross-examination as to the time at which the *first loss of weight*, if any, had begun.

It is in this inquiry that the greatest patience and caution are required; for the first answers given by patients to direct questions on the subject of loss of flesh and loss of weight

are almost invariably wrong and of the most misleading description. Seeing the great difficulty there is in getting at the truth in this matter, one cannot help the conviction that many delusive conclusions may have been arrived at based upon statements elicited without a sufficient knowledge of the necessity for caution on this point.

It will at once be recognised how very large a number of cases of hæmoptysis must have been rejected on these three counts.

4. I soon found that it was necessary to reject all females from the reports, and for the following reasons: (*a*) Their weights before admission could not be relied upon, the majority had never been weighed till they came to the hospital, and even if they had been weighed, the differences of clothing were found to present more unavoidable sources of fallacy than in men. (*b*) Pregnancy, childbearing, and lactation were constant elements of fallacy as to weight. (*c*) Hæmoptysis was found to be complicated with climacteric and other derangements of the menstrual functions to an extent seriously damaging its clinical meaning with relation to consumption. (*d*) In pushing a close inquiry into the history of a case, it was much more difficult to obtain a connected and reliable account of facts from the ***hospital class*** of women than from men of the same class. This rejection of all females from my inquiry was a serious loss, diminishing the number of eligible cases by about one half.

5. All cases were rejected in which there was reasonable suspicion of cardiac complications.

6. All cases were rejected who could not give a fairly succinct account of the onset of cough.

7. All cases were rejected who could not give an approximative estimate of the quantity and character of the expectorated blood in the first and subsequent hæmoptysis.

8. And, finally, after the inquiry had been completed, all cases were rejected if it was found on comparing the principal statements that they were inconsistent with one another.

It will be seen that the elements of these rigorous rejec-

tions are such that they do not give the cases a selected character in the sense of invalidating their claim to represent an *unprejudiced average* of cases of hæmoptysis occurring at a public hospital. They may, therefore, be considered to form a fairer basis for statistics than if no selection had been made,—having the great advantage that incomplete and unreliable reports are excluded.

It is evident that the labour of recording 100 cases was immensely enhanced by the above plan; but I think it will be felt that the value of the facts obtained has been proportionately increased.

If we allow for each case accepted one rejection on the 4th count (sex), two on the 3rd count (history of first loss of weight), two on the 2nd count (history of actual heaviest weight), and one between counts 6, 7, and 8 (and these numbers are below the truth), the 100 cases will be representative of an average succession of not less than 700 cases, in addition to the cases excluded under cardiac complications (count 5), and hæmoptysis in *streaks only* (count 1).

On examining the general table (Table 1)[1], it will be seen that each line in the horizontal direction represents a complete case. The primary object, as already stated, was to ascertain the true position of hæmoptysis in the etiology of consumption. But it was necessary to this end that a number of circumstances connected with each case should be recorded, which form a basis for a variety of other inquiries. It has been an especial object of my care, therefore, in arranging and analysing the records to place them in such a form that others can work from them just as well as myself. Indeed, it is chiefly with this view that I have wished to place the whole of the materials in the hands of the Society.

I regret that the work of collecting, tabulating, and analysing has occupied so much more time than I anticipated that it has left but little at my disposal for the purpose of reasoning and drawing conclusions from the results; and I must offer this as my apology to the Society

[1] The table has been found too large and intricate for publication (see Abridged Summary, pp. 266—270).

for presenting a paper which must, I fear, be tedious and uninteresting to hear, while the subjects which it offers for discussion are too numerous to be brought within the compass of a Society's meeting.

The following is an abridged summary of the most important facts contained in Table I, to which I have added seventeen supplementary tables,[1] in which some of the principal groups of facts are analysed and compared. Many other tables may yet be constructed with advantage to elucidate the meaning of other groups of facts contained in Table I, but for these I have not yet been able to find the time. On some future occasion I hope to be able to present a statement, in which the results of this investigation are compared with those obtained by Drs. Williams, Walshe, Niemeyer, and others—a work which I have already commenced.

[1] It has not been found practicable to publish these long and complicated tables.

Abridged Summary of Table I.

Columns 1 to 20.	The Table consists of an analysis of 100 cases of hæmoptysis in males, in all of which some disease existed in the upper lobes of the lungs at the time the note of the case was taken.
	The average age was 33·37 years.
	The condition of the lungs is classified in Table II (not published).
	The general symptoms were moderate in 43 per cent., severe in 41 per cent., extreme in 11 per cent.
	The average present weight of each patient was 122·97 pounds.
	The average heaviest weight (ever attained by each patient) was 142·62 pounds.
	The average present weight was therefore less than average heaviest weight by 19·65 pounds.
	In not one case was the present weight greater than the heaviest previous weight.
	The present weight was less than the former weight in every case.
	The heaviest weight had been entirely regained in 2 per cent.; but in each of these cases present weight was less than previous heaviest weight (see these cases, Cases 43, 97, with commentaries).[1]
	The average time elapsed since loss of weight began was 950·17 days.
	A cause for the loss of weight was assigned by the patient in 70 per cent.

[1] It has not been found practicable to publish the cases.

Abridged Summary of Table I—continued.

Columns 21 to 35.	Previous to admission of patient loss of weight had been treated with cod oil in 28 per cent., with pancreatic emulsion in 1 per cent., with oil and emulsion in 15 per cent., with a voyage in 1 per cent. No treatment had been adopted in 55 per cent.
	The average time elapsed since first cough began was 1464·4 days.
	A cause of first cough was assigned in 50 per cent.
	First cough began before first loss of weight in 69 per cent. „ „ after first loss of weight in 31 per cent. „ „ before first hæmoptysis in 87 per cent. „ „ after first hæmoptysis in 2 per cent. (These two cases, with commentaries, were given in detail, Cases, G. H., 26; R. W., 62. (Case 26, not published ; Case 62, published, p. 277.) „ „ At the same time as first hæmoptysis, *i. e.*, was accompanied by it, in 12 per cent. (These twelve cases were further analysed in Table III, not published.)
	The average time elapsed since present cough began (cough present at time of admission) was 915·45 days.
	The average time elapsed between the beginning of first cough and the beginning of present cough was 548·95 days.
	A cause for present cough was assigned in 50 per cent.
	Present cough began before loss of weight in 63 per cent. „ „ after loss of weight in 37 per cent. „ „ before first hæmoptysis in 77 per cent. „ „ after first hæmoptysis in 10 per cent. „ „ at the same time as first hæmoptysis in 13 per cent.

Abridged Summary of Table I—continued.

Columns 36 to 47.

The average time elapsed since the occurrence of *first* hæmoptysis in each case was 633·05 days. Comparing this with column 19, which shows that the average time elapsed since first loss of weight began was 950·17 days, it is seen that first loss of weight began on an average 317·12 days before first hæmoptysis.

But this general average is corrected in Tables IV, V, and VI (not published).

Table IV shows that in 8 cases loss of weight began on an average 853 days after first hæmoptysis.

Table V shows that in 82 cases loss of weight began on an average 469·78 days before first hæmoptysis.

Table VI shows that in 10 cases first loss of weight and first hæmoptysis were coetaneous.

The blood expectorated in first hæmoptysis was mixed in the sputa (flesh coloured) in 12 per cent.

Black or dark blood in 1 per cent. (In this case over ʒx in quantity, followed by smaller quantities on several occasions, produced by lifting.)

Florid blood in 76 per cent.

In small clots in 15 per cent.

In streaks in 9 per cent. (In each of these cases blood was subsequently expectorated in some other form.)

The quantity of blood expectorated in first hæmoptysis was under ʒss in twenty-four hours in 11 per cent.

Over	ʒss	„	34 per cent.
Over	ʒss	„	27 per cent.
Over	ʒx	„	26 per cent.

A cause for first hæmoptysis was assigned in 89 per cent.

First hæmoptysis occurred before the period of heaviest weight in 2 per cent. (Analyses of these cases were given, but are not published.)

After the period of heaviest weight in 87 per cent.

Abridged Summary of Table I—continued.

Columns 48 to 67.

First hæmoptysis occurred before first loss of weight in 8 per cent. (Analysed in Table IV, not published.)

„ „ after first loss of weight in 82 per cent. (Analysed in Table V, not published.)

„ „ at the same time as first loss of weight in 10 per cent. (Analysed in Table VI, not published.)

Hæmoptysis had recurred in 78 per cent.

First loss of weight occurred at the time of *recurrence* of hæmoptysis in 2 per cent. (These two cases were given in detail with commentaries, not published.)

Columns 52 to 65 give particulars of the quantity and quality and other details of recurrent hæmoptysis.

The family history shows that

Father only was consumptive in 9 per cent.

Mother „ „ 3 per cent.

Both father and mother were consumptive in 5 per cent.

Either one or both parents were consumptive in 17 per cent.

One brother (and no sister) was consumptive in 14 per cent.

One sister (and no brother) was consumptive in 7 per cent.

Three sisters (and no brother) were consumptive in 1 per cent.

One brother and one sister were consumptive in 4 per cent.

Two brothers and one sister were consumptive in 1 per cent.

Abridged Summary of Table I—continued.

Columns 68 to 73	Either brothers and sisters, or brothers or sisters, were consumptive in 26 per cent. One paternal uncle or aunt (no maternal) consumptive in 3 per cent. One maternal uncle or aunt (no paternal) consumptive in 0 per cent. One paternal and one maternal uncle or aunt consumptive in 1 per cent. Either paternal or maternal or both paternal and maternal uncles and aunts were consumptive in 4 per cent. In no case more than one uncle or one aunt on the same side consumptive. One paternal (and no maternal) first cousin consumptive in 1 per cent. (In no case more than one.) One maternal (and no paternal) first cousin consumptive in 1 per cent. (In no case more than one.) Both paternal and maternal first cousins consumptive in 0 per cent. Only one relative was consumptive in 19 per cent. Only two „ „ 7 per cent. Only three „ „ 7 per cent. Only four „ „ 1 per cent. In only one case were more than four relatives consumptive, this was Case 5, in which mother, one brother, three sisters, one first cousin (*paternal*), were consumptive. It is seen, therefore, that in the 100 cases there were 59 consumptive relatives, or, including the patients themselves, 159 consumptive individuals.

With reference to the important question of the true position of hæmoptysis in the natural history of pulmonary consumption, it will be observed that the 100 cases tabulated

in Table I arrange themselves into three great clinical groups, characterised by the relation in point of time between the first hæmoptysis and the first loss of weight.

And again into three other groups, characterised by the relation in point of time between the onset of first cough and the occurrence of first hæmoptysis.

And again into two groups, characterised by the relation in point of time between the onset of first cough and the onset of first loss of weight.

First hæmoptysis occurred before first loss of weight in 8 cases (8 per cent.). (Table IV, not published.)

— At the same time as first loss of weight in 10 cases (10 per cent.). (Table VI, not published.)

— After first loss of weight in 82 cases (82 per cent.). (Table V, not published.)

First cough occurred before first hæmoptysis in 87 cases (87 per cent.).

— At the same time as first hæmoptysis in 12 cases (12 per cent.). (Table III, not published.)

— After first hæmoptysis in 2 cases (2 per cent.). (See abridged summary of Table I, p. 267.)

First cough occurred before first loss of weight in 69 cases (69 per cent.).

— After first loss of weight in 31 cases (31 per cent.).

I have taken loss of weight as the most palpable sign of constitutional decline, and Column 16, Table I, shows that in every one of the 100 cases the patient weighed less than he had done at a previous period of his life; and a comparison of Columns 14 and 15, Table I, shows that the average of weight per patient was 19·65 pounds. The rate at which this loss had occurred in the different groups of cases is shown in Table VIII (Table not published, abstract given below[1]):

[1] In 82 cases in which loss of weight began *before* first hæmoptysis—Rate about 7 pounds per 365 days.

In 10 cases in which loss of weight began at *same time* as first hæmoptysis—Rate about 8 pounds per 365 days.

In 8 cases in which loss of weight began *after* first hæmoptysis—Rate about 11 pounds per 365 days.

in Table IX (not published) I have shown the total loss of weight and the rate at which loss of weight had occurred at the different stages of lung disease.

Column 18, Table 1 (not published), shows that in only two cases out of the 100 had the *previous heaviest weight* been entirely regained at any time since the first onset of disease; and in these two the recovery of weight was altogether exceptional in its circumstances and meaning, as shown in the commentaries on the cases (not published).

Column 28, Table I, shows that in 12 cases cough began at the same time as first hæmoptysis, and I have constructed a special Table for this group of cases in which the leading facts of each can be seen at a glance (Table III, not published).

It is a striking fact that 82 *of the* 100 *patients had lost weight before the occurrence of the first hæmoptysis*—the average time during which this loss had taken place previous to the hæmorrhage being no less than 469·7 days. It is then self-evident that in these 82 cases some diseased condition existed before the occurrence of hæmorrhage, and that, therefore, we have no reason for seeking a cause in the hæmoptysis.

Our special interest is, therefore, concentrated upon the 18 cases forming the two smaller groups arranged in Tables IV and VI (not published).

In Table IV it is shown that first hæmoptysis preceded first loss of weight by 853 days on an average in 8 cases.

In Table VI first hæmoptysis and first loss of weight were coetaneous in 10 cases.

In either of these groups, therefore, it is clearly possible to urge that the hæmoptysis was the cause of the constitutional decline, and of all the local disease accompanying it.

To each of these 18 cases, therefore, I have devoted a special commentary, with a view to clearing up as far as possible the position of the hæmoptysis as a cause of disease; and these commentaries may be examined immediately.[1]

[1] It has only been practicable to publish Cases 62, 17, 58, 42, 93, and their commentaries.

First, however, it is necessary to point out that one clinical fact, in addition to hæmoptysis and loss of weight, at once assumes a right to prominence in discussing these cases. I mean the *onset of the first cough*[1] and its relation in point of time to the occurrence of first hæmoptysis. I have therefore given it a special place in Tables IV and VI. For as cough must be taken to indicate some defective condition of the respiratory organs, if cough preceded first hæmoptysis by any appreciable period of time, it throws at once a grave doubt upon the claim of the hæmorrhage to be regarded as the first cause of the lung disease.

Now we have seen by Table I that out of the 100 cases first cough preceded first hæmoptysis in 87, and began after the first hæmoptysis in only 2.

And turning to the 18 cases now more particularly under consideration, it is seen, by Table IV, that first cough preceded first hæmoptysis by 924 days on an average in 7 out of the 8 cases, and thus indicated that some kind of mischief was going on in the respiratory tract before bleeding occurred. In the one case out of the 8 (Case 62) in which the cough began 84 days after the first hæmoptysis the circumstances were peculiar, and are fully discussed in the commentary to the case (see p. 278).

In Table VI it is seen that in 6 out of the 10 cases cough preceded first hæmoptysis by 1600 days on an average, the minimum being 197 days. In the other four cases the cough, hæmoptysis, and loss of weight *began at the same time.* These were Cases 17 (p. 279), 42 (p. 282), 58 (p. 284). In each of these cases it will be seen that there is room for the conclusion that the lung disease proceeded from the hæmorrhage, although in neither of them is this conclusion inevitable. And the same may be said of Case 15 (not published), Table IV; in which first hæmoptysis *preceded* loss of weight. The commentaries on these cases therefore claim

[1] In recording "first cough" care was taken only to take note under that head when some connection could be reasonably traced between the cough and the illness connected with first hæmoptysis.

our special consideration. But in Case 93 (p. 285) our interest culminates, for in it there appears to be no room for any more rational conclusion than that the lung disease was produced by the damage done by the hæmorrhage (see p. 275, par. *d*).

Among the many subjects of interest contained in Table I, for which I have not time in this paper, I may mention one intimately connected with the cases herein discussed. I mean the question, whether the results of pulmonary hæmorrhage bear any relation, *in respect to tissue damage*, to the *quantity* of blood expectorated in hæmoptysis. The materials for a separate table representing the facts relating to this question are to be found in Table I; and at some future day I hope to deal with them. In this place I will only say that I am disposed to think that it is of more importance whether the hæmorrhage has taken place *into the respiratory tubes*, or *into the pulmonary tissues*, than whether it has been profuse or slight. Analogy lends weight to this opinion, as seen in cerebral hæmorrhage, and other instances of bleeding into the tissues, and into the passages of internal parts.

It will be observed that throughout this paper, and in the commentaries on the cases, I have studiously avoided entering upon questions of pathological histology and the use of debateable terms, my object being to keep as strictly as possible to a simple analysis of the clinical facts presented by the cases produced.

In concluding, however, I may just venture to enumerate what appear to me to be some of the principal heads under which hæmoptysis may be arranged in the natural history of pulmonary consumption, as justified by clinical observation.

As a symptom it may be thus classified—

1. In a large number of cases it is simply the result of congestion and disintegration of a highly vascular organ, in the course of a disease of constitutional origin.

2. In a large number of cases it is simply the result of congestion and disintegration of a highly vascular organ in the course of diseases of local origin.

3. In a certain number of cases it is simply the result of accidents temporarily overdistending the vascular system of the lungs, and leading to their rupture; in the same way as similar overdistension leads to rupture of vessels in other parts of the body.[1]

4. In a certain number of cases it is the result of the bursting of small aneurisms in the lungs formed in the course of lung disease.

As a cause of lung disease and constitutional decline—

I am disposed to place it only as one item, and that a very occasional one, in a large and important group, embracing *all foreign substances which find their way into the perivascular* and *perialveolar tissue* of the lungs, and by their irritation there, set up lymphatic (adenoid) and connective tissue cell-proliferation and its consequences.

Of this important group the following are some of the principal constituents:

a. The dust of flint, coal, iron, and other substances inhaled by workers in different dusty trades.

b. The products of inflammatory destruction of tissue.

c. The products of catarrhal affections.

d. The débris of blood, and of tissues disintegrated by the extravasation of blood (see Case 93, referred to, p. 274).

e. Albuminoid tissue disintegrated by peroxidation, in true tuberculosis.[2]

[1] Whether such overdistension is competent to cause rupture of vessels, the walls of which are not previously diseased, is a very wide question, which must be argued with reference to the whole vascular system before it is specialised within the narrower area of the pulmonary circulation. Is it necessary to assume that a vessel was diseased because it burst under exceptional distension?

[2] In my opinion the disintegrated albuminoid tissue is the irritant which

Lastly, hæmoptysis may possibly become a cause of lung disease by accumulation of blood débris in the alveoli (see Case 93, p. 285).

sets up that hyperplasia of adenoid tissue and its results, so well described by Portal, Virchow, Sanderson, and Rindfleisch. But whereas they place this hyperplasia first among the pathological changes of tuberculosis, I give precedence to the peroxidation and disintegration of albuminoid tissue, of which the hyperplastic changes are but the effects, the order of events being, according to my view—

a. Deficiency of fat in the blood.

b. Peroxidation of albuminoid tissue.

c. The production of disintegrated albuminoid tissue, the result of peroxidation.

d. Hyperplasia of adenoid tissue, the result of irritation of the absorbent system engaged in removing the disintegrated tissue.

Whether the disintegrated albuminoid tissue, or the resulting diseased adenoid tissue shall be called "tubercle" is of little consequence, so that the distinction in the order of events is borne in mind.

From the analysis of 8 *cases, in which first hæmoptysis occurred before first loss of weight.* (Table IV, not published.)[1]

Case 62.	Male.
R. W—.	Age 27.
	Right lung, softening Left lung, softening } upper parts.
	General symptoms extreme.
	Present weight, 111 pounds.
	Heaviest weight, 131 pounds.
	Present weight less than heaviest weight by 20 pounds.
	Time elapsed since first loss of weight, 252 days. 28 days after first hæmoptysis.
	Cause of first loss of weight assigned, *nil.*
	Time elapsed since first cough began, 196 days. 84 days after first hæmoptysis.
	First cough began *after first* loss of weight, 56 days.
	„ „ after first hæmoptysis, 84 days.
	Present cough began at the same time as first cough (it had never ceased).
	Time elapsed since first hæmoptysis, 280 days. 28 days before first loss of weight.
	The blood of first hæmoptysis was florid.
	The blood of first hæmoptysis was in quantity over ℥x in 24 hours.
	The cause of first hæmoptysis assigned was "dancing and drinking iced ale."
	First hæmoptysis occurred before first loss of [illegible] 28 *days.*
	Hæmoptysis did not recur.
	The family history was free from consumption.

[1] This is one of two cases in which first cough began after first hæm[illegible] (pp. 267, 273).

Commentary.—This is a typical case of consumption proceeding *primâ facie* entirely from hæmoptysis. Nothing could be extracted by the most careful inquiry to account for the hæmorrhage, except the cause assigned by the patient, viz., that "while at his full weight and in usual health he overheated himself with dancing, and then drank some iced ale, and immediately broke a blood-vessel in his chest." No recurrence of hæmorrhage had taken place. Loss of weight began about a month after the accident, and twenty pounds had been lost in 252 days. *No cough set in till* 84 *days* after the accident and 56 days after the loss of weight, but since it began it had never ceased. This is a remarkable circumstance, for if the decline in health, indicated by *loss of weight, were due to local disease set up by the hæmorrhage*, it is very improbable that it should not have excited the ordinary symptom of lung disease or irritation, viz., cough.

When we regard these facts in relation with the condition of the lungs on admission, *i. e.*, softening of both upper lobes, it becomes still more remarkable that no cough should have been excited by the hæmorrhage, if it be supposed that the structure of both lungs was so much damaged by the effusion of blood as to lead to all that followed. My opinion is that these circumstances throw the greatest doubt upon such a supposition, and I am led to put aside the *primâ facie* conclusion that the whole case started from the hæmorrhage, and to conclude, instead, that the following is a much more probable solution:—That at the time the accident occurred the patient was not in such unquestionable health as he imagined; that his habits of dancing, drinking, and late hours were telling upon his constitution; that his subsequent decline was imminent at the time of the accidental hæmorrhage; that the hæmorrhage brought on by the temporary overheating and excitement, instead of causing disease of the lungs, *protected* them; that the relief to the vascular system, combined with the rest and ease enforced by the alarm caused by the hæmorrhage, actually postponed those changes in the lung which, otherwise, might have appeared much sooner than they did;

and that thus it is that we have a history of twenty-eight days in which no obvious change took place in the health—then constitutional decline and loss of flesh for fifty-six days, before sufficient irritation had occurred in the lungs to cause a cough. If then we put aside the hæmorrhage, as accidental, we see an ordinary case of consumption in which the events follow the usual order:

1. Damaged constitutional health (by fast living?).
2. Decline of weight and strength.
3. Lung irritation and cough.
4. Advancing lung disease and constitutional decline.

From the analysis of 10 *cases, in which first hæmoptysis and first loss of weight occurred at the same time.* (Table VI, not published.)

Case 17.	Male.
A. G—.	Age 31.
	Right lung normal Left lung consolidated } upper parts.
	General symptoms moderate.
	Present weight, 116 pounds.
	Heaviest weight, 128 pounds.
	Present weight less than former weight by 12 pounds.
	Time elapsed since first loss of weight, 1460 days. Same time as first hæmoptysis.
	Cause of first loss of weight assigned, hæmoptysis.
	Loss of weight was not treated with either oil or emulsion.
	Time elapsed since first cough began, 1460 days. It was accompanied by first hæmoptysis.
	Cause of first cough assigned, *nil.*
	First cough began before (just before) loss of weight.

First cough began before, or at the same time as, first hæmoptysis.

Present cough began at the same time as first cough.

Time elapsed since first hæmoptysis, 1460 days; 1458 days before last hæmoptysis; 1108 days before first *profuse* hæmoptysis.

The blood of first hæmoptysis was florid.

The quantity of blood in first hæmoptysis was over ʒss in 24 hours.

Cause of first hæmoptysis assigned, *nil.*

First hæmoptysis occurred after heaviest weight.

„ „ at the same time as first loss of weight.

Hæmoptysis had recurred.

Time elapsed since last hæmoptysis, 2 days. 250 days after first profuse hæmoptysis; 1458 days after first hæmoptysis and first loss of weight.

The blood of last hæmoptysis was florid.

The quantity of blood in last hæmoptysis was over ʒx in 24 hours.

Last hæmoptysis occurred after first loss of weight. 1458 days.

The father was consumptive.

The mother was consumptive.

One brother was consumptive.

Commentary.—Without assignable cause, hæmoptysis to the extent of ʒss in twenty-four hours occurred with cough 1460 days ago, and was immediately followed by loss of weight. Cough had continued ever since.

Eleven hundred and eight days after the first hæmorrhage a second and much more profuse bleeding occurred, and 230 days later a third and very profuse hæmoptysis took place.

The only lung disease detected by physical examination was consolidation of the left upper lobe. The total loss of weight was twelve pounds in 1460 days. Consumption existed in both parents, and also in collaterals.

Seeing that the hæmorrhage occurred without any external cause, that 1460 days had elapsed since the first bleeding, and 252 days since the last, and that no other lung disease than consolidation existed, it would hardly be rational to consider the constitutional decline as due to this amount of disease, and therefore irrational to look upon the hæmorrhage as the cause of both the lung disease and loss of weight.

If the statement that no loss of weight had occurred up to the date of first hæmoptysis were correct, we are without palpable evidence of constitutional decline as a cause of lung disease prior to the hæmorrhage; but the strong consumptive history in the family and the absence of external cause of hæmorrhage make it most probable that some hereditary defect existed. And as it appeared afterwards that the heaviest weight by weighing was taken two years before the first hæmoptysis, it is open to question whether he had not unconsciously lost weight in that time. He was weighed at the hospital at the time of the second hæmoptysis 250 days before he was weighed again (two days after the last bleeding); and on both of the occasions his weight was the same, but as he quickly gained four pounds under treatment, it showed that this weight had been a morbidly low one. Although, therefore, the case is obscure, there is no clear evidence that the hæmorrhage caused the lung disease.

From the analysis of 10 *cases, in which first hæmoptysis and first loss of weight occurred at the same time.* (Table VI.)

Case 42.	Male.
R. C—.	Age 41.
	Right lung consolidated and softening Left lung normal } upper parts.
	General symptoms moderate.
	Present weight, 143 pounds.
	Heaviest weight, 159 pounds.
	Present weight less than heaviest weight by 16 pounds.
	Time elapsed since first loss of weight, 504 days. Same time as first hæmoptysis.
	Cause of first loss of weight assigned, hæmoptysis from overlifting.
	Loss of weight had not been treated with either oil or emulsion.
	Time elapsed since first cough began, 504 days. Same time as first hæmoptysis and first loss of weight.
	Cause of first cough assigned, overlifting.
	First cough began before loss of weight (just before). "Coughed and brought up blood, and then lost weight."
	First cough began at the same time as first hæmoptysis.
	Present cough began at the same time, and was assigned to the same cause as first cough.
	Time elapsed since first hæmoptysis, 504 days.
	The blood of first hæmoptysis was florid.
	The quantity of blood in first hæmoptysis was over ʒss in 24 hours.
	The cause of first hæmoptysis assigned was cough from overlifting.
	First hæmoptysis occurred at the same time as first loss of weight (*i. e.*, loss of weight dated from coughing up the blood).

	Hæmoptysis had recurred.
	Time elapsed since last hæmoptysis, 84 days. 420 days after first hæmoptysis.
	The blood of last hæmoptysis was florid.
	The quantity of blood in last hæmoptysis was over ʒss in 24 hours.
	Family history free from consumption.

Commentary.—He was certain he was in usual health and usual weight (159 pounds) 504 days ago when he overlifted, coughed, and "broke a blood-vessel," and he had never been well since. In six weeks' treatment with oil and emulsion at the Royal Hospital he gained six pounds, but was still ten pounds below his average weight. Sixteen pounds weight had been lost in 504 days, commencing with first hæmoptysis and first cough. The cough is tabulated as beginning before hæmoptysis, because it was stated to have *caused* the hæmorrhage, but as no considerable interval occurred between the two—the cough coming on and hæmoptysis immediately following—it is quite possible that internal bleeding was the cause of the cough, instead of *vice versâ*.

The hæmoptysis was over ʒss in twenty-four hours, and was assigned to cough produced by overlifting. Hæmoptysis had recurred; the last attack being over ʒss in twenty-four hours 84 days ago.

There was no consumption in the family, and apparently dated from the accident. The case is fairly open to the opinion that the *lung disea up by the hæmorrhage*, although that conclus inevitable.

From the analysis of 10 *cases in which first hæmoptysis and first loss of weight occurred at the same time.* (Table VI.)

Case 58.	Male.
A. B—.	Age 22.
	Right lung softening } upper parts. Left lung excavated }
	General symptoms extreme.
	Present weight, 99 pounds.
	Heaviest weight, 118 pounds.
	Present weight less than heaviest weight by 19 pounds.
	Time elapsed since first loss of weight, 1095 days. (Same time as first hæmoptysis; same time as first cough; same time as present cough; 1088 days before last hæmoptysis.)
	Cause of first loss of weight assigned, *nil.*
	Loss of weight had not been treated with either oil or emulsion.
	Time elapsed since first cough began, 1095 days.
	Cause of first cough assigned, *nil.*
	First cough began before first loss of weight (just before).
	First cough began before first hæmoptysis (just before). (Cough was said to cause hæmoptysis, and therefore preceded it by a fraction of time.)
	Present cough began at same time as first cough.
	Time elapsed since first hæmoptysis, 1095 days.
	The blood of first hæmoptysis was florid.
	The quantity of blood in first hæmoptysis was over ℥x in 24 hours.
	Cause of first hæmoptysis assigned, cough.
	First hæmoptysis occurred at the same time as first loss of weight.

	Hæmoptysis had recurred.
	Time elapsed since last hæmoptysis, 7 days.
	The blood of last hæmoptysis was mixed with sputa (flesh-coloured sputa).
	The quantity of blood in last hæmoptysis was under ʒss.
	Last hæmoptysis occurred after first loss of weight. 1088 days.
	One brother was consumptive.

Commentary.—It was just before first hæmoptysis that he weighed the heaviest weight known (118 pounds), and he was certain no loss of weight had taken place up to that time, but he had been growing, and was only about 19 years old. One brother was consumptive, and as the break down (consisting in cough hæmoptysis and loss of weight without apparent cause) occurred just about the age of completion of growth, it is most probable that the symptoms were simply coëtaneous indications of the hereditary disease assuming an active stage. It is, however, open to discussion whether the hæmorrhage caused the subsequent changes in the lungs. The first hæmoptysis was over ʒx in twenty-four hours.

From the analysis of 10 *cases in which first hæmoptysis and first loss of weight occurred at the same time.* (Table VI.)

Case 93.	Male.
A. B—.	Age 28.
	Right lung consolidated } upper lobes. Left lung softening }
	General symptoms severe.

Present weight, 112 pounds.

Heaviest weight, 149 pounds.

Present weight less than heaviest weight by 37 pounds.

Time elapsed since first loss of weight, 365 days. Just after, or at same time as, first hæmoptysis; same time as, or just after, first cough.

Cause of first loss of weight assigned, hæmoptysis.

Loss of weight had been treated with oil and emulsion.

Time elapsed since first cough began, 365 days.

Cause of first cough assigned, hæmoptysis. Cough accompanied and was said to cause hæmoptysis.

First cough began before, or at the same time as, first loss of weight.

First cough began before, or at the same time as, first hæmoptysis.

Present cough began at the same time, and was due to same cause as first cough.

Time elapsed since first hæmoptysis, 365 days.

The blood of first hæmoptysis was florid.

The quantity of blood in first hæmoptysis was over ʒx in 24 hours.

The cause of first hæmoptysis assigned was overlifting a heavy weight.

First hæmoptysis occurred at the same time as first loss of weight.

Hæmoptysis had recurred.

Time elapsed since last hæmoptysis, 56 days. 309 days after first hæmoptysis.

The blood of last hæmoptysis was florid.

The quantity of last hæmoptysis was over ʒss in 24 hours.

Last hæmoptysis occurred after first loss of weight, 309 days.

The family history was free from consumption.

Commentary.—After six weeks' oil and emulsion he gained five pounds, making his weight 117 pounds = 32 pounds less than heaviest weight. The heaviest weight known was three months before first hæmoptysis, and he declared there was no loss of weight or sign of illness up to the moment of his lifting a heavy weight, when he began to cough up blood profusely. The first hæmoptysis was said to be three pints of florid blood in twenty-four hours. From that time up to two months ago frequent smaller bleedings had occurred. Right lung very slightly consolidated; left, dull and chronically disintegrating, not very active.

In this case no consumption was known in the family, and everything appeared to date from an accidental strain in lifting a very heavy weight, which was immediately followed by extremely profuse hæmoptysis. An enormous loss of weight had taken place—thirty-seven pounds (a quarter of his total heaviest weight) in 365 days—in spite of treatment; and smaller bleedings had frequently occurred. One lung was consolidated, and the other slowly softening.

Under oil and emulsion he gained five pounds, but was still thirty-two pounds short of his normal weight.

This case approaches more nearly than any other out of the 100 here analysed to the description of Niemeyer's "galloping consumption, of which bronchial hæmorrhage is the immediate cause;" but even here there is a history of 365 days, and the patient was still alive and beginning to gain flesh after his great loss, whereas Niemeyer speaks of such cases perishing in a few months.

This is the only case out of the 100 in which the most rational conclusion appears to be that the lung disease was caused by the hæmorrhage. (See pp. 273, 275, 276.)

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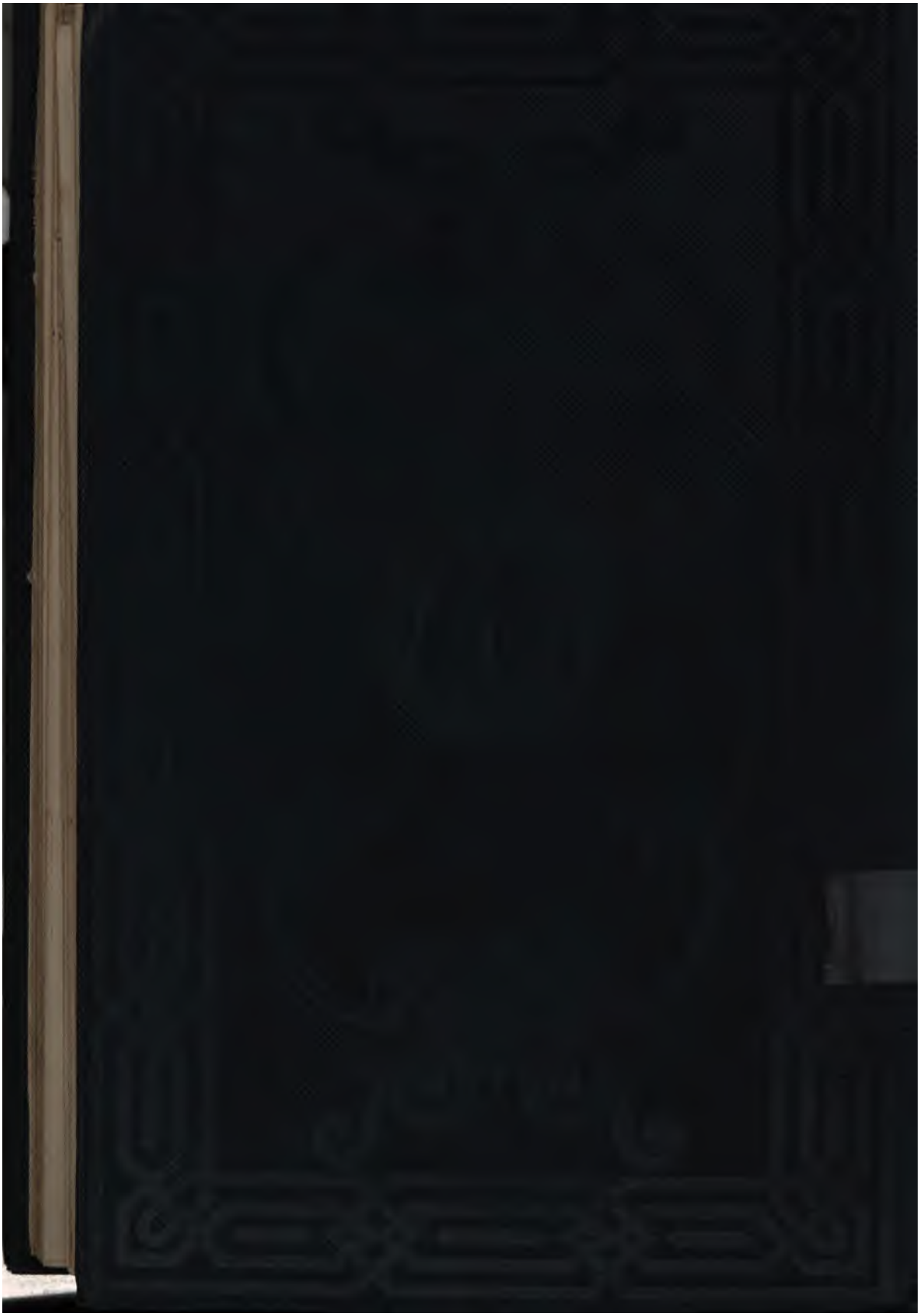
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